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PSYCHOSOMATIC SYMPTOMS RESULTING FROM THE IMPACT OF WAR

Observations in Civilian Medical Practice

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SINCE society has become conscious of and concerned with the health of its people, it has recognized that war acts as an extremely deleterious agent. War produces lack of food, lack of shelter, lack of clothing, lack of medicine and lack of hygiene. War's effect on health, therefore, is generally expressed in terms of these physical causes.

Only since the first World War has any scientific interest been taken in the effect of the emotional disturbances of war on the physical well-being of the people involved. Even so, only superficial attention was paid to this aspect of war. Generally, when emotional disturbances were mentioned, it was to explain the causes of war and the causes of aberrations in behavior, either of the individual person, or of a social, economic, civil, racial or religious mass or group. But that the emotions may exert a profound influence on the physical health of human beings is a concept which has received wide acceptance only within relatively recent years.

The influence of the emotions on physical health has long been recognized, but it was accepted only in certain esoteric fields, such as magic, superstition, religion, mesmerism and charlatanism. There were a few insightful physicians, however, who preached the importance of the emotions in relation to physical health, but, until recently, they were practically unheard. This is readily explainable when one realizes that the medical profession was going through a miraculous period of materialism which promised to reveal all the secrets of the soma. So intriguing was the illumination which the various sciences threw on the workings of the human body that it was forgotten that, besides a soma capable of astounding processes of manufac-

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ture, control, maintenance and dissolution, there is also always a psyche—that patients are people as well as material bodies.

Recently, as a result of the discoveries regarding the unconscious, the interrelationship between the psyche and the soma began to be uncovered. As a result, a discipline called psychosomatic medicine has arisen which is directed toward a study of these interrelationships.

From these studies it has become obvious that war can produce deterioration not only through physical insults but also through emotional insults. Emotional insults influence not only the behavior of groups and persons but their bodily health.

PSYCHOSOMATIC EFFECTS ON PERSONALITY

The more obvious physical disabilities appearing in the course and wake of war are those associated with the soldier himself. Many soldiers and potential soldiers display physical disturbances generated by the emotions. These often legitimately prevent their entering into, or lead to their discharge from, the service. Such disabilities are the product usually of old emotional patterns that exist in the unconscious, brought to the surface by the stresses of the emergent situation of war. On the other hand, certain persons are apt to improve in physical health on going into the service. Both these reactions may occur in the so-called passive-dependent person. The passive-dependent person who in civil life is living in a protective environment shows physical symptoms to avoid service. The passive-dependent person living in a hostile environment often finds that the well ordered existence which is run for him in the service gives him a sense of security that he does not have at home.

ALLERGY AS A PSYCHOSOMATIC MANIFESTATION

The two manifestations described were particularly well shown during and after the war in the so-called allergic group of persons. Many of these, free of bronchial asthma since early childhood, experienced a recurrence of their difficulty on notification that they were to be inducted into service; or they had a recurrence when they found life in the service not to their liking. It must be emphasized, of course, that these reactions were entirely unconscious and not to be classified as malingering. The asthmatic patient is generally, from a psychologic point of view, a passive-dependent person, and, therefore, both types of reactions were to be anticipated. Time after time in practice it was found that the asthmatic person who had been suffering practically to the day of his entry into service remained free of symptoms throughout the period of his enlistment, with episodes of asthma only when on leave and visiting his former home. Many of these

veterans on discharge resumed their asthmatic pattern as it had been before their entrance into the service. The condition in the other group who acquired asthma at the threat of induction or under stress while in the service cleared up completely on return to civil life, unless circumstances, whether economic, social or psychologic, still presented a problem which challenged their passive dependency.

Another group of persons who had allergic symptoms during the war were the wives of men called away to service. In many instances again, two opposite patterns presented themselves. In one portion of the group, the absence of the husband apparently spelled insecurity, and symptoms were then precipitated. In another portion, the departure of the husband spelled a degree of independence which had been wished for but had been lacking. These women often lost symptoms which had been present before their husbands left. This was a common reaction among female allergic patients, who are often of the dominating type as defense against their own passive dependency.

PSYCHOSOMATIC MANIFESTATIONS OF ALLERGY IN CHILDREN

As regards children, two main problems present themselves to the physician. The first has to do with separation of the child from the father or the mother or both, and the second with the return of the father or mother or both.

A group of children which lends itself well to analysis of the emotional dynamics of separation and return on physical well-being is the allergic group. These children present a characteristic pattern of emotional disturbance which leads to the development, aggravation and prolongation of so-called allergic symptoms, of which asthma is perhaps the most striking example. As has been said, psychiatrists have classified allergic persons, and particularly asthmatic persons, as passive dependents. By individual analysis of both adults and children, this passive dependency appears to be based on a need for the mother. The persistence of this pattern in adult asthmatic persons is quite evident. The origin of the pattern, however, lies in childhood, as does the implication of the characterization of the asthmatic attack as a cry for the rejecting mother. It is then actually the rejecting mother who provides the emotional basis for the asthmatic manifestation. This observation, founded on psychoanalytic technic, has been further confirmed by our work with allergic children.

Recently, we observed a group of allergic children in comparison with a group of nonallergic children. The allergic children had been identified as such both from the usual medical history and physical examination and from the cutaneous tests for allergic agents which gave positive reactions in all cases. Psychologic examinations in the form of interviews or diagnostic play sessions were held with each

child, and interviews were had with the mother in every instance and in many instances with the father also. A group of children of comparable age and economic background brought to the office for personal behavior problems was used as a control. None of the latter children, on physcial examination or medical history, gave any indication of allergic symptoms. The interviews with the parents indicated that the children in the allergic group had been much more commonly rejected by the mother than those in the nonallergic group. The criterion of rejection used in this study was defined as follows: Verbal expression of rejection on the part of the mother whose behavior toward the child is such that she consciously or unconsciously has a desire to be free from the child and considers it a burden. Under this criterion, maternal rejection, and by the same token longing for the mother or, in David Levy's term, affect hunger, appears to be an almost universal experience of the allergic child.

PSYCHOSOMATIC EFFECTS OF MATERNAL REJECTION IN WARTIME

During the war and since, maternal rejection and the threat of maternal rejection, as will be shown, were considerably enhanced. As a result, many examples of children suffering from the somatic effects of this emotional trauma were seen in the physicians' and, especially, the allergists' offices. These effects could often be traced to the factors aforementioned, viz., the departure of the father or mother or both and their return.

The departure of the father apparently created or increased the feeling of insecurity in the mother. This often aggravated or provoked a sense of rejection on the part of the child, producing, precipitating or aggravating his allergic symptoms. The departure of the mother to the factory appears to have been an even more direct manifestation of maternal rejection to the child. Children also suffered from a sense of rejection from the constant moving about of the family which was so common during the war. Often, this meant longer or shorter periods of strain and tenseness on the part of the mother which inevitably reflected themselves in varying degrees of neglect of the child's feelings.

One of the most interesting phenomena presented was the product of the return of the father after long absence—often after the child had lost all conscious memory of him, or, as in the case of a child born after his departure, held no remembrance of him at all. His return then represented a threat to the child by presenting a rival for the mother's affection. With the feeling of rejection often already present, this event precipitated attacks of allergic symptoms that were extremely difficult to clear, for the hostility of the child often created hostility in the returning parent and so increased the actual rejection.

The mother returning from work which had kept her away from the child for a good part of the day to a situation in which she was almost constantly with him gave the child opportunity to utilize his allergic symptoms to punish her for her previous neglect of him. This use of allergic symptoms to express hostility, or perhaps to mask the feelings of guilt and anxiety which hostility engenders, is a common phenomenon which we have previously reported.[1]

The following case report is characteristic of the psychosomatic dynamics involved in the production of symptoms in an allergic child under the stresses which the war placed on his family. It also points up the obvious fact before mentioned, that in most instances the emotional pattern of the person who breaks under the strain of the war and its sequelae has been previously present. The war serves only to accentuate or bring to critical issue the burdens on the psyche.

REPORT OF A CASE

A 5 year old allergic boy, whose mother had entered into a marriage of convenience which proved to be incompatible, from infancy had had chronic hay fever. It was evident the mother rejected the child, probably since his birth. When her husband left for military service, she began an extramarital affair and turned the child over almost completely to the care of a nurse. The child had his first attack of asthma when the mother learned that the father was returning from foreign service. She threw herself more deeply into her extramarital affair and utilized the nurse to keep the child out of the way. Evading his care was only one sign of the rejection, which she voiced in such phrases as "I can't be tied down by him"; "He drives me crazy"; "I've a life of my own to lead, after all."

She masked her rejection, however, by being oversolicitous about his physical care, his feeding and his sleeping, taking him to many physicians and attending him in his numerous attacks of asthma. Such oversolicitousness and overprotection are common characteristics of mothers of allergic children. It is probably a reaction formation to the feeling of guilt over rejection of the child and takes the form generally of overattentiveness to the child's physical needs with neglect of his emotional needs.

This child's yearning for affection showed up in a story that he told the therapeutist.

"Once there was a little boy who didn't want his mother to leave him. He said, 'Please, mother, don't leave me.' His mother said, 'If you be good I won't leave you. If you be bad I will leave you.' So the little boy was naughty, but not very, though. So the mother ran outside the house and got very quietly in the car and started the car and zoomed away. So he ran to the neighbor and said, 'Will you take care of me?' And the neighbor said, 'No.' So he ran to another neighbor and asked, 'Will you take care of me?' And the neighbor said, 'Yes, we've been looking for a little boy.' So they did and they loved him lots."

After the father's return, the mother dismissed the nurse. But she maintained the habit of leaving the child furtively without telling him. She would, for

1. Miller, H., and Baruch, D. W.: Psychological Dynamics in Allergic Patients as Shown in Group and Individual Therapy, J. Consult. Psychol. **12**:111, 1948.

instance, sneak out of the waiting room while the child was in his play therapy session.

One day, looking out of the window, he saw her drive away. He cried and cried, utterly miserable, "I want her; I want her; why did she leave me?" on and on. Then, finally, he turned on the therapeutist. "If you don't get my mother back," he said, "I'll cough. I'll get asthma. Then she'll have to come."

When she returned, he asked tearfully, "Why did you leave me?" She said, "I had to pick up a package. Don't be angry." He answered, "I wasn't angry at you, mommy. I love you. I love you lots. I was angry at Dorothy (the therapeutist). I'm never angry at you but I hate Dorothy."

This episode was one example of how the child was apparently using his asthma to secure greater contact and closeness with the mother. It showed also that he used it as a kind of threat or as a weapon against her. It was clear from the protocols that he felt hostile to his mother but blocked the direct expression of such feelings for fear of driving her further away. He could, however, make her uncomfortable through his asthma and, at one and the same time, hide the underlying motive from sight. In short, he used his asthma to gain closeness, to express hostility and to disguise his hostility in such a way as to let himself escape from too great a burden of feeling guilty.

CONCLUSION

The impact of war and its aftermaths are often evident in disturbances of physical well-being. These disturbances are not necessarily on the basis of deprivation of food, shelter, clothing and other material needs but on deprivation of those essential emotional needs, such as love, appreciation, understanding and acceptance. The allergic child provides a particularly good example for interpretation of the dynamics involved, because war and its aftermaths aggravate the underlying maternal rejection which stimulates the appearance of the somatic symptoms of allergy.

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STREPTOMYCIN THERAPY IN TUBERCULOUS MENINGITIS

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IN ATTEMPTING to evaluate my results with streptomycin, I shall take into consideration the immediate clinical response, the subsequent course of the disease, the frequency of complications, the occurrence of relapses and the length of time the patient remained alive.

In addition to the study of the effects of streptomycin, I shall report on the cerebrospinal fluid changes, before, during and after treatment and also on the streptomycin levels in the blood and cerebrospinal fluid.

I should like to call particular attention to the fact that, with the exception of 1 patient who received streptomycin intrathecally for nine days, all these patients received streptomycin intramuscularly only.

I treated 19 patients, whose ages ranged from 6 months to 13 years. There were 10 boys and 9 girls; 15 were Negro, 3 Mexican and 1 white. The preponderance of Negroes among this series may be explained by the fact that a large number of the patients at the Cook County Hospital are Negro.

The dosage employed in this series was 200,000 units given intramuscularly every three hours. This treatment was continued for ninety days.

RESULTS

At the present writing (May 23, 1948), 8 of the 19 patients are still alive. One has been alive for fifteen months, 1 for thirteen months, 1 for twelve months, 1 for eleven and one-half months, 1 for eight months, 1 for five months, and 2 for three months from the beginning of treatment (table 1).

The immediate response to streptomycin was usually striking. Patients who entered the hospital in a lethargic state, sometimes even moribund, became alert within a few days. In several children, the temperature dropped immediately; in others, it receded after several days. I learned, however, not to be too enthusiastic about the early favorable response. Patients who appeared to be doing well for a long time suddenly took a turn for the worse and died. In spite of the

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fatal outcome, however, streptomycin evidently did succeed in prolonging their lives. Seven of the 11 patients who died lived for more than seven weeks and 1 for almost seven months after the beginning of treatment (table 2). Only 4 of these patients lived less than two weeks. (table 3). The prolongation of life by streptomycin becomes doubly apparent when one compares the results obtained since the advent of streptomycin with those before the discovery of this antibiotic substance.

Isaacson and I [1] reviewed a large series of cases at Cook County Hospital before the streptomycin era. We discovered that most of the

TABLE 1.—*Data on Patients with Tuberculous Meningitis Who Improved on Administration of Streptomycin*

Name	Age	Color	Sex	Date of Admission	Streptomycin		Duration of Treatment, Days	Results (5/23/48)	Complications
					Intramuscularly	Intrathecally			
A. B.	10 mo.	N	M	2/23/47	200,000 units every 3 hr.	100,000 units daily for 9 days	90	Living; 15 mo.	None
K. P.	21 mo.	N	M	4/20/47	200,000 units every 3 hr.	None	90	Living; 13 mo.	None
C. L.	1 yr.	N	M	5/19/47	200,000 units every 3 hr.	None	90	Living; 12 mo.	Deafness
F. B.	8 yr.	N	M	6/ 6/47	200,000 units every 3 hr.	None	90	Living; 11½ mo.	None
M. A. H.	8 yr.	W	F	9/24/47	200,000 units every 3 hr.	None	90	Living; 8 mo.	Right hemiplegia; blindness with optic atrophy; paralysis of left phrenic nerve
L. S.	4 yr.	N	F	12/23/47	200,000 units every 3 hr.	None	90	Living; 5 mo.	None
J. R.	2½ yr.	N	F	2/12/48	200,000 units every 3 hr.	None	90	Living; 3 mo.	None
C. C.	5 yr.	W	M	2/19/48	200,000 units every 3 hr.	None	90	Living; 3 mo.	None

patients at that time died within two to three weeks after their admission. None lived more than seven weeks after the onset of the meningeal symptoms.

A review of the present series of patients throws some light on the pathogenesis of tuberculous meningitis.

All but 1 of these patients showed miliary tuberculosis on roentgenogram or at autopsy. The exception was a child with tuberculous meningitis following tuberculosis of the hip. Tuberculoma of the brain was observed only once, in a child who had generalized tuberculosis. I believe that tuberculous meningitis is nearly always a part of a

1. Unpublished data.

TABLE 2.—*Data on Patients Treated with Streptomycin Who Lived More Than Seven Weeks and Then Died*

Name	Age	Color	Sex	Date of Admission	Streptomycin		Duration of Treatment, Days	Duration of Life, Days
					Intramuscularly	Intrathecally		
M. W.	21 mo.	N	M	10/22/47	200,000 units every 3 hr.	None	90	200
D. L.	4 yr.	N	F	9/22/47	200,000 units every 3 hr.	None	90	189
P. H.	9 yr.	N	M	12/11/47	200,000 units every 3 hr.	None	90	155
J. G.	8 yr.	W	M	1/ 9/48	200,000 units every 3 hr.	None	90	97
P. B.	10 mo.	N	F	1/12/48	200,000 units every 3 hr.	None	86	86
E. H.	4 yr.	N	M	9/ 1/47	200,000 units every 3 hr.	None	79	79
E. A.	2 yr.	N	F	2/ 7/48	200,000 units every 3 hr.	None	50	50

TABLE 3.—*Data on Patients Treated with Streptomycin Who Died in Less Than Seven Weeks After Onset of the Meningeal Signs*

Name	Age	Color	Sex	Date of Admission	Streptomycin		Duration of Treatment, Days
					Intramuscularly	Intrathecally	
W. S.	14 mo.	N	M	2/13/47	200,000 units every 3 hr.	None	24
P. G.	4 yr.	W	F	1/26/48	200,000 units every 3 hr.	None	16
J. R.	13 yr.	N	F	4/ 8/47	200,000 units every 3 hr.	None	13
B. G.	16 mo.	N	F	1/ 3/48	200,000 units every 3 hr.	None	9

TABLE 4.—*Duration of Illness in Children with Tuberculous Meningitis Before the Streptomycin Era*

Duration of Illness, Days	Number of Patients
Under 5	71
6 to 10	76
11 to 20	79
21 to 30	13
31 to 49	7

hematogenous spread, and not an extension from a tuberculoma as postulated by Rich and McCurdock.[1a]

Clinically, the distinguishing feature in the present series was the frequency of encephalitic symptoms, of ocular disturbances and of abdominal pain.

Encephalitic phenomena accompanied and often overshadowed the meningitic symptoms in 17 of the 19 patients. The encephalitic sign noted most frequently at the onset of the disease was irritability. Next in frequency were somnolence and convulsions. Only 2 of the children were alert throughout the course of the disease.

Paralysis of ocular muscles was a part of the encephalitic phenomena manifesting themselves early in the disease. Among these were ptosis, internal strabismus and inequality of the pupils.

Hemiplegia occurred early in the disease in 2 children and persisted for a long time. Paralysis of the facial nerve accompanied the hemiplegia but subsided with residual asymmetry.

The frequency of encephalitic symptoms corroborates my opinion that tuberculous meningitis is a meningoencephalitis.[2]

Among the meningitic signs, nuchal rigidity was the first to appear. Brudzinski and Kernig signs were next; the appearance of the Babinski reflex was inconstant. Of course, all meningitic signs are inconstant or entirely absent in young infants.[3]

Seven of the patients were admitted with a history of abdominal pain. This symptom was responsible for a wrong diagnosis in several instances. Because of the predominance of abdominal symptoms, the admitting diagnosis for 1 child was intestinal obstruction and for another, peritonitis.

COMPLICATIONS AND SEQUELAE

It is difficult to decide which symptoms are a part of the original disease and which constitute a complication. It is also hard to determine whether the complications are due to the meningitis or to the streptomycin.

In addition to the hemiplegia, which was discussed under symptoms, paraplegia occurred in 1 child five months after entrance, when the meningeal symptoms had subsided and the patient was afebrile. The

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1a. Rich, A., and McCurdock, M.: An Enquiry Concerning the Role of Allergy, Immunity and Other Factors in the Pathogenesis of Human Tuberculosis, Bull. Johns Hopkins Hosp. **44**:274, 1929; Pathogenesis of Tuberculous Meningitis, ibid **52**:5, 1933.

2. Levinson, A.: Meningitis Masked by Encephalitic Symptoms, Arch. Pediat. **49**:6, 1932.

3. Levinson, A.: Meningitis, in Brennemann, J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Company, Inc., 1942, vol. 4, chap. 8.

paralysis could not be attributed to intrathecal administration of streptomycin as the patient had received the drug intramuscularly only.

One patient who had hemiplegia and paralysis of the facial nerve also had paralysis of the left phrenic nerve. One child had choroiditis. There were 3 with papilledema and 1 with optic atrophy. Two of the patients became deaf. The question that naturally arose was whether the deafness was due to the streptomycin or to the meningitis, as it is a well known fact that any form of meningitis may produce deafness. One of the patients entered the hospital with deafness, so it was known positively that streptomycin could not have been the causative factor. In the other patient the cause was a matter of conjecture.

Vestibular disturbances occurred in 4 patients but cleared up in all of them. One patient had a maculopapular rash that lasted two days. This rash was undoubtedly due to the streptomycin. One child had hematuria that cleared up within four days. One had peritonitis and herpes zoster. Another had bursitis of the left knee. The bursa contained tubercle bacilli. Still another had tuberculous osteomyelitis of the right tibia.

Three patients acquired mumps; 5 had measles and 4, chickenpox. The intercurrent infections did not seem to affect their recovery from the original disease.

RELAPSES

Up to date, 7 patients have had relapses. Two recovered from the relapse on the resumption of streptomycin administration and 5 died. The relapses were characterized by encephalitic symptoms, mainly irritability, vomiting and lethargy. Meningitic symptoms were absent or mild. With the encephalitic symptoms, there was also a miliary involvement of the chest that showed up roentgenologically. One patient also had bilateral cervical adenopathy which subsided within three days after the readministration of streptomycin. I was surprised to find that 4 of the patients who had relapses were afebrile with no reduction in the glucose level of the cerebrospinal fluid.

LABORATORY DATA

Roentgenologic Appearance.—With the exception of the single child mentioned previously in whom the meningitis followed a tuberculous hip disease, the roentgenograms of the chest all showed tuberculous involvement of the lungs with miliary or tuberculous pneumonia. The pathologic involvement of the chest regressed with the improvement of the patient and ran parallel to the cerebrospinal fluid changes. The roentgenologic changes continued for a long time after the meningeal signs subsided but finally cleared up in the patients who improved.

Blood.—In the majority of patients, the leukocytes averaged 10,000 per cubic millimeter, with only a moderate increase even at the height of the disease. In 1 patient only did the leukocyte count reach 22,000. In most instances, there was a relative increase in the polymorphonuclear forms at first, with a gradual change to lymphocytosis. In 1 child, the eosinophils were as high as 21 per cent. In other children, they ranged from 5 to 11 per cent. The eosinophilia usually appeared during the third month of the streptomycin treatment.

In 1 patient, the red blood cell count rose beyond 6,000,000 per cubic millimeter, reaching 6,640,000 at one time. In another child, the red cell count exceeded 6,000,000 only once. The mechanism of this phenomenon is hard to explain. I do know, however, that it occurs in some infections of the central nervous system.

The sedimentation rate in the patients who were followed up was elevated on entrance. It usually ranged between 24 and 32 mm. per hour. It subsided gradually and became normal when the patient was clinically well.

Urine.—One patient had hematuria which cleared up in four days. Another showed sugar in his urine once, but he had had glucose intravenously that day. With the exception of the one with hematuria, none of these patients showed any albumin in the urine.

Tuberculin Tests.—The reaction to the tuberculin test was positive in all the children. In 1, however, it was negative at the beginning but became positive later with the improvement of the patient. In 2 children, the reaction was cachectic.

Cerebrospinal Fluid.—The most interesting and most illuminating laboratory data for these patients were in connection with the cerebrospinal fluid changes. For the first time in the history of the disease, the gradual recession of the pathologic changes of the cerebrospinal fluid with the improvement of the patient could be watched. Before evaluating the changes that the cerebrospinal fluid undergoes in persons treated with streptomycin, one must keep in mind the fact that even though these changes follow a certain pattern they are not always uniform. The only certain way to study the effect of streptomycin on the cerebrospinal fluid is to observe the fluid of the patient at various intervals.

The most outstanding changes noted in the cerebrospinal fluid in these patients were the occurrence of xanthochromic fluid, the variations in cell count and protein curve after therapy and the rapid increase in glucose content during streptomycin therapy.

Xanthochromia was present in 5 children, although the time of its appearance and persistence varied. In 1 child, it was present on admission but cleared up two weeks after treatment with streptomycin

was started and remained clear. In another child who had xanthochromia on admission, the condition has persisted to the present—five months later. A third child had clear fluid on his admission but two weeks later xanthochromia developed, lasted for five months and then cleared up. This patient is doing well. Another patient was admitted with a clear fluid but two months later xanthochromia became evident and persisted for three months. The fluid then became colorless, but the patient died two weeks later. In the fifth child, the xanthochromia appeared during a relapse which was followed by death in ten days.

Xanthochromia in these children evidently was not due to the streptomycin. I believe it was due to the minute hemorrhages of the cord and to stasis of the cerebrospinal fluid caused by the interference with its normal circulation. Strange to say, next to brain tumor, xanthochromia occurs most frequently in tuberculous meningitis and not in purulent meningitis.

The cerebrospinal fluid cell count varied. When the fluid was clear or opalescent, it ranged from 65 to 450 cells per cubic millimeter, with an average of 250. When the fluid was xanthochromic, the cell count was high, in 1 child as high as 1,700 per cubic millimeter. After the administration of streptomycin, the cell count usually rose for the first ten to fourteen days. Then it gradually fell, even in those who did not survive. In patients who recovered, it often took from three to three and one-half months for the cells to reach normal. However, when the fluid was xanthochromic, the cell count remained high until the fluid cleared up.

The protein was always increased on admission. When the fluid was clear or opalescent, it ranged from 75 to 275 mg. per hundred cubic centimeters of fluid. It was exceptionally high, however, in all 5 patients with xanthochromia. In 1 of these, it was 3,000 mg. per hundred cubic centimeters. The protein level usually rose for about a month and then gradually tapered off to normal in four to five months, if the patient recovered. The chloride content was low and, as a rule, remained below the normal level of 700 mg. per hundred cubic centimeters for some time.

The reaction to the Levinson test was in line with the rest of the cerebrospinal fluid changes. It was positive on admission and remained so for two to three months, gradually becoming less positive and finally becoming negative.

Most surprising was the effect of streptomycin on the glucose content of the cerebrospinal fluid. The glucose level was always reduced on admission, rising a day or two after the beginning of the streptomycin treatment. It then became reduced again and gradually returned to normal. In 1 child, it returned to normal in less than a week and

in 8 children, in less than a month. The increase in glucose is an indication of a favorable immediate response but is not an index of the final prognosis. The normal glucose level during a relapse can be explained on the ground that the relapse is due to tuberculous encephalitis instead of meningitis.[4]

Streptomycin Levels.—Streptomycin in various amounts was obtained from the blood and cerebrospinal fluid. The specimens of blood and cerebrospinal fluid were taken approximately three hours after the last injection of streptomycin and just before the next injection. The streptomycin level of the blood ranged from 0 to 160 units, in most instances between 10 to 40 units. In 3 patients, no streptomycin was obtained in the blood at the first testing but was detected in fairly large amounts the next time.

The streptomycin level in the cerebrospinal fluid ranged from 0 to 320 units, with 5 to 20 units as the average (table 5).

As a rule, the streptomycin level was lower in the cerebrospinal fluid than in the blood. In 3 patients, however, it was the same in both, and in 5 it was higher in the cerebrospinal fluid than in the blood. This last phenomenon can be explained by the fact that in the patients mentioned the streptomycin had already left the blood and entered the cerebrospinal fluid. Similar results were obtained when glucose was administered to dogs. After a certain lapse of time, the sugar level receded in the blood and rose in the cerebrospinal fluid.

The level of streptomycin in the blood and cerebrospinal fluid was no criterion of the recovery of the patient. Death ensued in several instances in which the streptomycin level was high. The streptomycin levels did, however, indicate that the antibiotic substance had penetrated into the cerebrospinal fluid.

COMMENT

It is still too early to state that streptomycin produces complete recovery in tuberculous meningitis. Some of the patients reported as recovered or clinically well either died later or became mentally deficient. Lincoln, Kirmse and DeVito [5] stated that of the 80 patients of Debré, 50 per cent died and not more than 5 per cent of those who survived were in good clinical remission. The patient of Cooke, Dunphy and Blake [6] who survived for thirty months after streptomycin

4. Cohn, D. J.; Levinson, A., and McCarthy, F.: Physiological Variations in the Glucose Ratio of Blood and Cerebrospinal Fluid, Am. J. Physiol. **103**:613, 1933.

5. Lincoln, E. M.; Kirmse, T. W., and DeVito, E.: Tuberculous Meningitis, J. A. M. A. **136**:593 (Feb. 28) 1948.

6. Cooke, R. E.; Dunphy, D. L., and Blake, F. G.: Streptomycin in Tuberculous Meningitis: A Report of Its Use in a One Year Old Infant, Yale J. Biol. & Med. **18**:221, 1946.

was started became mentally retarded and probably deaf. Krafchick's [7] patient, on the other hand, twenty-one months after treatment was begun and twelve months after the relapse, is still alive and his mentality and development are normal. My experience is similar to that

TABLE 5.—*Streptomycin Levels in Children with Tuberculous Meningitis Treated with Streptomycin Intramuscularly*

Name	Date	Amount of Streptomycin, Units	Interval, Hours	Streptomycin Levels Units/Cc.	
				Blood	Cerebrospinal Fluid
A. B.	4/ 2/47	200,000	3	20	0
	4/14/47	200,000	3	40	40
	4/28/47	200,000	3	160	5
K. P.	4/28/47	200,000	3	5	10
	5/ 9/47	200,000	3	40	20
	7/22/47	200,000	3	20	2.5
C. L.	5/25/47	200,000	3	20	5
	6/16/47	200,000	3	10	..
	7/ 9/47	200,000	3	..	0.62
	7/22/47	200,000	3	20	10
F. B	6/16/47	200,000	3	10	..
	7/ 9/47	200,000	3	..	0.62
	7/22/47	200,000	3	20	2.5
D. L.	10/ 2/47	200,000	3	2.5	1.25
	10/30/47	200,000	3	5	2.5
	12/ 2/47	200,000	3	0	1.25
E. H.	10/ 2/47	200,000	3	10	320
	10/30/47	200,000	3	1.25	20
M. A. H.	10/ 2/47	200,000	3	0	5
	12/16/47	200,000	3	20	10
M. W.	11/ 4/47	200,000	3	2.5	0
	12/ 2/47	200,000	3	5	0
	1/ 5/47	200,000	3	..	10
P. H.	1/15/48	200,000	3	10	..
	2/25/48	200,000	3	40	20
L. S.	1/20/48	200,000	3	20	20
	2/25/48	200,000	3	40	40
J. G.	1/27/48	200,000	3	20	5
	3/ 3/48	200,000	3	0	10
	4/ 8/48	200,000	3	0	0
P. B.	1/27/48	100,000	3	0	0
	3/ 4/48	100,000	3	10	2.5
	3/25/48	100,000	3	10	5
C. C.	3/23/48	200,000	3	40	20
	4/23/48	300,000	6	2.5	1.25
J. R.	4/23/48	300,000	6	2.5	2.5
R. P.	5/14/48	300,000	6	5	20
M. A. H.	5/21/48	300,000	6	8	2

of the other investigators. Seven of the present patients who had been doing well suddenly had a relapse and died.

It is evident, however, that streptomycin prolongs life in tuberculous meningitis. The 8 patients who are still alive, 1 for fifteen months after the onset of the disease, testify to this fact. Even the 7 who died lived

7. Krafchik, L. L.: Tuberculous Meningitis Treated with Streptomycin, J. A. M. A. **132**:375 (Oct. 19) 1946.

far beyond the seven week period, the maximum time limit reached by the patient with tuberculous meningitis in the prestreptomycin era. Whether the addition of promizole® [5] (4, 2′-diaminophenyl-5′-thiazolyl-sulfone) will result in complete recovery, further clinical investigation will tell.

The dosage of streptomycin is far from established. The fourth Streptomycin Conference, held in St. Louis in 1947,[8] recommended 2 Gm. or more of streptomycin intramuscularly per day, in addition to 50 mg. intrathecally every day or two, for acute miliary and tuberculous meningitis.

From my experience with meningitis, I am convinced that intrathecal injections are unnecessary. With the dosage of 200,000 units every three hours, I have had good results in a number of cases. I am now following a new series which is being treated with 300,000 units of streptomycin every six hours. However, insufficient time has elapsed for a definite statement to be made.

Another question still to be settled is: Over how long a period should one continue to give streptomycin?

I gave it for ninety days with gratifying results in many cases. I did not feel that the duration of the treatment was in any way responsible for the complications several patients showed. I believe that they were most likely a consequence of the meningitis and not of the streptomycin. The deafness in 1 patient was definitely due to the meningitis, as it occurred before streptomycin was administered.

One of the problems that arose in connection with streptomycin therapy was what to do with the patient who was clinically well but whose cerebrospinal fluid continued to contain tubercle bacilli. This happened to 1 child six months after his admission. The patient was well and the cytologic and chemical findings in the cerebrospinal fluid were normal. However, since he was streptomycin resistant, the question arose whether he was to receive more streptomycin. It was decided not to give him any more of the drug. The patient remained well except for deafness, which he had previously.

Another question calls for deliberation: When should patients who are apparently well be sent home? I made it my policy to keep patients in the hospital until all meningeal symptoms had subsided and the chest was normal, clinically and roentgenologically. Then, if there were no other children in the family and if the adults were free from tuberculosis, the patient was permitted to go home.

How a relapse should be treated was another question that came up. I started administration of streptomycin again in all patients with relapse, and several of them improved and stayed well.

8. Dosage of Streptomycin in Tuberculosis, Current Comment, J. A. M. A. **135**:842 (Nov. 29) 1947.

REPORT OF CASES

A few typical cases in this series illustrate the results. Three of these children are still alive, 1 died after six and one-half months and 1 died after three months.

CASE 1.—A. B., a 10 month old Negro boy, was admitted to the Children's Division of the Cook County Hospital on Feb. 23, 1947, with a history of increasing

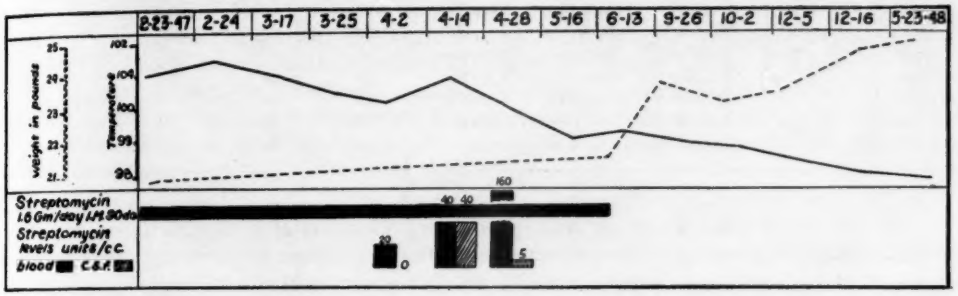

Fig. 1.—Graphic presentation of data on patient A. B. In the upper portion the solid line shows the curve for temperature and the dotted line the curve for weight. The bars in the lower portion show dosage and levels in the blood and cerebrospinal fluid of the streptomycin.

TABLE 6.—*Results of Laboratory Studies of the Blood of Patient A. B.*

Date	Hemoglobin, Gm./%	Red Blood Cells, Millions	Color Index	White Blood Cells	Neutrophils, %	Lymphocytes, %	Monocytes, %	Eosinophils, %	Basophils, %	Tuberculin Test	Roentgenographic Appearance
2/24/47	8.35/62	4.06	0.77	19,200	74	21	2	1	0	Mantoux (+) (1: 10,000)	Miliary tuberculosis
3/25/47	11.5/69	4.20	0.70	12,900	66	29	3	2	0	.....	.....
4/ 2/47	.....	....	....	.....	..	..	..	..	..	.....	Miliary tuberculosis
4/14/47	10/67	4.36	0.69	8,750	62	30	7	1	0	.....	.....
5/16/47	11.5/69	4.01	0.86	7,400	37	51	8	3	0	.....	.....
6/13/47	11.2/65	4.98	0.76	8,150	41	34	7	18	0	.....	Healing
9/26/47	11.5/69	4.52	0.74	11,200	27	48	4	21	0	Vollmer (+)	.....
10/ 2/47	11.5/69	4.74	0.73	17,500	36	42	12	8	1	.....	Negative
12/ 5/47	10/60	4.17	0.73	13,160	25	66	4	3	1	Mantoux (+) (1: 10,000)	....
12/16/47	10.5	4.84	....	11,700	47	36	11	6	0	Negative	.....
1/ 8/48	10.5/63	4.84	0.65	11,700	47	36	11	3	1	.....	.....
1/26/48	10/60	4.21	....	8,150	41	45	8	6	0	.....	.....

somnolence for four days and restlessness and vomiting for one day. There was a history of contact with a person with active tuberculosis in the same apartment.

The physical examination on his admission revealed decided lethargy, left internal strabismus, pronounced nuchal rigidity, bulging fontanel and the presence of the Kernig and Brudzinski signs. Fluoroscopy revealed a triangular area of increased density extending from the hilus of the right lung downward. The temperature was 101 F. (rectum). The diagnosis on admission was tuberculous meningitis.

The patient received 200,000 units of streptomycin intramuscularly every three hours for ninety days and 100,000 units intrathecally once a day for nine days.

TABLE 7.—*Results of Laboratory Studies of the Cerebrospinal Fluid of Patient A. B.*

Date	Appearance	Cells (Lymphocytes)	Smear	Cultures	Guinea Pig Reaction	Glucose, Mg./100 Cc.	Proteins, Mg./100 Cc.	Chlorides, Mg./100 Cc.	Tryptophane Reaction	Levin Reac
2/23/47	Clear	65	Negative	Negative	.....	22	180	560	.....	....
2/24/47	Opalescent	460	Negative	Positive	.....	58	90	600	.....	....
3/17/47	Clear	225	Negative	Negative	Negative	37	110	...	.....	Positi
3/25/47	Clear	335	Negative	Positive	.....	36	320	...	.....	....
4/ 2/47	Opalescent	346	.....	.....	.....	44	220	600	Positive	....
4/14/47	Clear	185	Negative	Negative	Negative	48	155	...	.....	....
4/28/47	Clear	220	Negative	Negative	.....	44	130	620	.....	....
5/16/47	Clear	133	Negative	Negative	Negative	42	95	676	Positive	Negati
6/13/47	Clear	26	Negative	Negative	Negative	50	40	...	Positive	Negati
9/26/47	Clear	6	Negative	Negative	Negative	72	56	...	Negative	Negati
12/16/47	Clear	1	Negative	Negative	Negative	74	36	...	Negative	Negati
4/26/48	Clear	8	Negative	Negative	.....	74	40	...	Negative	Negati

TABLE 8.—*Results of Laboratory Studies of the Blood of Patient C. L.*

Date	Hemoglobin, Gm./%	Red Blood Cells, Millions	Color Index	White Blood Cells	Neutrophils, %	Lymphocytes, %	Monocytes, %	Eosinophils, %	Basophils, %	Tuberculin Test	Roentgenographic Appearance
5/23/47	9.35/62	5.23	0.60	8,350	56	33	9	1	0	Mantoux (+) (1: 10,000)	Primary tuberculo
6/ 3/47	9/54	4.62	0.58	4,950	59	32	7	0	1	.....	.....
6/30/47	11.0	4.77	....	7,200	28	62	6	4	6	.....	Hilar N
7/22/47	10.5/63	4.35	0.73	4,850	35	50	11	4	0	.....	Clearing
8/ 1/47	13/78	5.21	0.75	6,400	30	60	5	5	0	Mantoux (+) (1: 1,000)	Regressi .....
9/26/47	12.5/75	4.80	0.78	7,300	45	52	0	2	0	Vollmer Patch (+)	Negative
10/22/47	12.5/75	4.40	0.90	7,750	38	50	7	4	1	Mantoux (+) (1: 10,000)	Negative .....
11/15/47	10.5/63	3.79	0.85	6,550	30	61	8	1	0	.....	.....
12/ 2/47	12/75	5.07	0.72	11,850	59	35	6	0	0	.....	Negative
1/ 9/48	12.5/75	4.19	0.91	7,600	19	70	8	2	1	.....	.....
2/ 7/48	10.5/63	5.26	0.69	8,900	20	67	10	2	1	.....	.....
3/ 9/48	11/66	4.98	0.68	10,800	32	61	1	1	0	.....	Negative
5/23/48	12/72	4.51	0.80	7,100	27	71	1	1	0	.....	.....

TABLE 9.—*Results of Laboratory Studies of the Cerebrospinal Fluid of Patient C. L.*

Date	Appearance	Cells (Lymphocytes)	Smear	Cultures	Guinea Pig Reaction	Glucose, Mg./100 Cc.	Protein, Mg./100 Cc.	Chlorides, Mg./100 Cc.	Tryptophane Reaction	Levin Reac
5/23/47	Xanthochromic	1,070	Negative	Negative	.....	38	...	...	.....	....
5/24/47	Xanthochromic	1,852	.....	.....	.....	..	...	...	Positive	Positi
5/27/47	Xanthochromic	1,730	Negative	Negative	Negative	72	670	...	.....	....
6/ 3/47	Xanthochromic	848	Negative	Negative	.....	62	560	...	Positive	Negati
6/ 9/47	Opalescent	932	Negative	Negative	.....	85	400	...	.....	....
6/16/47	Opalescent	783	Positive	Positive	.....	70	610	...	.....	....
6/30/47	Clear	....	Negative	Negative	.....	70	580	656	.....	....
7/ 9/47	Clear	510	Negative	Negative	.....	74	340	617	.....	....
7/22/47	Clear	280	Negative	Negative	.....	42	160	660	.....	....
8/ 1/47	Clear	93	Negative	Negative	.....	67	122	645	.....	....
9/26/47	Clear	40	Negative	Negative	Negative	70	40	...	Negative	....
10/22/47	Clear	53	Negative	Positive	.....	50	36	686	.....	....
12/ 2/47	Clear	40	Negative	Positive	Negative	55	72	728	Negative	....
3/ 9/48	Clear	3	Negative	Negative	Negative	74	46	749	Negative	Negati

The clinical picture changed from extreme lethargy to restlessness and irritability, which gradually decreased in severity. On completion of streptomycin therapy on May 23, three months after his admission, the patient appeared bright and interested in his surroundings.

The patient remained afebrile after the first few days, except for an infection of the upper respiratory tract that responded to sulfonamide and for attacks of measles and chickenpox.

At the present writing, at the end of fifteen months, the patient is doing well (fig. 1, tables 6 and 7).

CASE 2.—C. L., a 1 year old Negro boy, was admitted to the Children's Division of the Cook County Hospital, May 19, 1947, with a history of loss of weight (8 pounds [3.6 Kg.] in three weeks), nausea, vomiting and constipation for ten days. The mother reported that the child had several colds during the winter but was alert and playful when vomiting began. Constipation required enemas and cathartics for relief.

Delivery was normal. Artificial feeding had been used with adequate cod liver oil and orange juice. Development was normal with no illness except infections of the upper respiratory tract. The mother was treated for syphilis during pregnancy. There was no history of tuberculosis in the family or of an exposure to the disease.

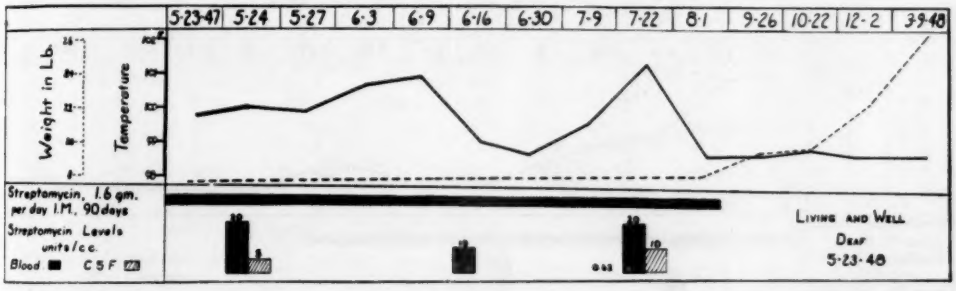

Fig. 2.—Graphic presentation of data on patient C. L. In the upper portion the solid line represents the curve for temperature and the dotted line the curve for weight. The bars in the lower portion show dosage and levels in the blood and cerebrospinal fluid of the streptomycin.

Physical examination on his admission revealed a pale, asthenic, lethargic infant with some nuchal rigidity and slight mucoid discharge from the nose. The heart, lungs, abdomen and reflexes were normal.

The first impression on admission was anemia of sickle cell, infectious or secondary type, to rule out a low grade intermittent intestinal obstruction. After a spinal puncture, the diagnosis was changed to tuberculous meningitis.

The patient was started on streptomycin therapy, 200,000 units every three hours for ninety days. No streptomycin was given intrathecally. The meningeal signs gradually subsided, although the patient remained irritable for some time. On July 7, less than two months after treatment was started, the patient was discovered to be hard of hearing. On July 26, three weeks later, the patient had measles. The course of the measles was uneventful.

At the present writing, the patient is afebrile, alert and happy. Hearing, however, has not returned (fig. 2, tables 8 and 9).

CASE 3.—F. B., an 8 year old Negro boy, was admitted June 6, 1947, with a history of weakness and inability to walk for one day. The mother stated that the

child had been perfectly well until he had a slight cough. Three days before his admission he complained of headache and pain in the side. He also complained of hearing difficulty and loss of appetite. He became dyspneic.

On June 4, two days prior to entrance, he was unable to walk because of weakness. The speech impediment the child had had become worse.

The child had had measles and chickenpox. The father died of heart disease. The mother was living and well. There was no history of exposure to tuberculosis.

On his admission the patient's temperature was 99.8 F., the pulse rate 68 and respirations 18.

He had rigidity of the neck and positive Kernig and Brudzinski signs. He lay on his right side with knees drawn up and the head in mild extension. Bilateral punch tenderness over the abdomen and tenderness over the last lumbar vertebrae were present. The right leg was weaker than the left. Deep tendon reflexes were present and equally active. Cremasteric and abdominal reflexes were not elicited.

The diagnosis was tuberculous meningitis to rule out poliomyelitis, acute radiculitis and tuberculosis of vertebra (Pott's disease). Administration of streptomycin, 200,000 units every three hours intramuscularly, was started June 6 and continued for ninety days.

The patient continued to be apathetic with decided rigidity of the neck, strong Kernig and Brudzinski reflexes and stiffness of the back.

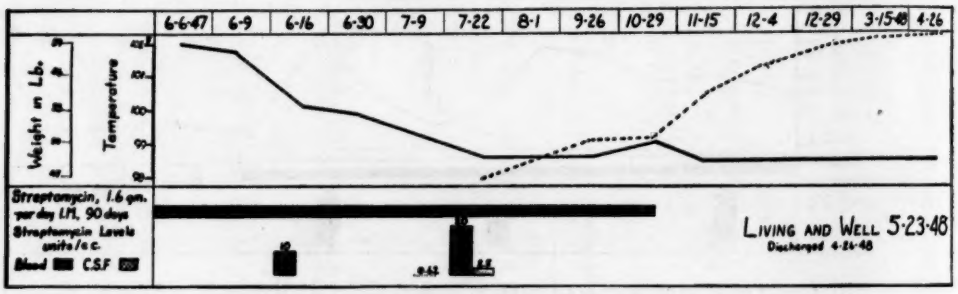

Fig. 3.—Graphic presentation of data on patient F. B. In the upper portion the solid line represents the curve for temperature and the dotted line the curve for weight. The bars in the lower portion show dosage and levels in the blood and cerebrospinal fluid of the streptomycin.

On June 11, the patient became well oriented. The Kernig and Brudzinski signs, however, were still present. Within a week, the patient began to improve and sat up and walked with assistance. Hearing was deficient for a few weeks and then improved.

About five months after his admission the patient showed weakness in the legs. He was unable to stand or walk and could sit up only occasionally. The patellar reflex on the left side was absent. Ankle clonus developed. He also had small ulcers on his right knee and upper part of the back. The hearing improved considerably. His mentality was good.

On December 5, a month later, he began to walk with some assistance. The left patellar reflex returned and was even exaggerated. Walking improved gradually.

At the present writing, the patient is well and hearing is normal (fig. 3, tables 10 and 11).

At first, the question was whether the deafness was due to streptomycin. However, the history of deafness four days prior to the administration of streptomycin points to the original meningoencephalitis as the cause.

The paralysis of the lower extremities is difficult to account for. The patient received no streptomycin intrathecally, so no local reaction to streptomycin can be considered. The paralysis was possibly due to radiculitis or to hemorrhage into the subarachnoid space secondary to tuberculous meningitis.

TABLE 10.—*Results of Laboratory Studies on the Blood of Patient F. B.*

Hemoglobin, Gm./%	Red Blood Cells, Millions	Color Index	White Blood Cells	Neutrophils, %	Lymphocytes, %	Eosinophils, %	Monocytes, %	Basophils, %	Tuberculin Test	Roentgenographic Appearance
11.2/75	4.61	0.81	11,200	74	12	0	14	0	.....	.....
.....	....	....	.....	..	..	..	..	..	Mantoux (negative) (1: 100,000)	.....
.....	....	....	14,000	68	15	5	9	0	Mantoux (+) (1: 10,000)	Miliary tuberculosis
13.5/81	4.86	0.81	7,800	46	41	3	8	2	.....	Miliary tuberculosis
13.5/81	4.97	0.81	7,300	61	31	5	1	2	.....	Miliary tuberculosis
11.5/69	4.74	0.73	10,100	36	50	5	7	2	Vollmer Patch (+)	Clearing
13.5/81	4.55	0.90	8,150	49	42	6	3	0	Mantoux (+) (1: 10,000)	.....
13.5	3.91	....	7,600	32	56	5	6	1	.....	Healed miliary tuberculosis
14.5/87	4.54	0.95	7,650	34	50	11	5	0	.....	.....
13.0	4.77	....	7,900	37	52	2	8	0	.....	Negative
12/72	4.40	0.81	8,960	48	37	8	7	0	.....	.....
12.5/75	4.31	0.87	9,550	40	58	1	1	0	.....	.....
12.5/75	5.06	0.75	13,300	30	63	4	1	2	.....	Negative
12/72	4.30	0.75	10,000	45	44	1	10	0	.....	Negative

TABLE 11.—*Results of Laboratory Studies on the Cerebrospinal Fluid of Patient F. B.*

Appearance	Cells (Lymphocytes)	Smear	Cultures	Guinea Pig Reaction	Glucose Mg./100 Cc.	Protein, Mg./100 Cc.	Chlorides, Mg./100 Cc.	Tryptophane Reaction	Levinson Reaction
Clear	240	Negative	Positive	Positive	32	155	687	Positive	Positive
Clear	275	Positive	Negative	.....	25	350	620	.....	.....
Xanthochromic	449	Negative	Negative	.....	87	Quantity not stated	...	.....	.....
Xanthochromic	...	Positive	Positive	.....	117	2,750	...	.....	.....
Xanthochromic	256	Positive	Positive	.....	72	1,700	560	.....	.....
Xanthochromic	75	Positive	Negative	.....	50	740	666	.....	.....
Xanthochromic	81	Negative	Negative	.....	82	800	666	.....	.....
Xanthochromic	120	Negative	Negative	.....	95	1,600	676	Positive	.....
Xanthochromic	59	Negative	Negative	.....	85	1,900	645	Negative	Negative
Clear	22	Negative	Negative	Negative	72	60	666	Negative	Negative

The xanthochromia can be explained as the result of a local pathologic involvement in the cord or around it, possibly by hemorrhage into the spinal subarachnoid space.

CASE 4.—M. W., a Negro boy 22 months of age, was admitted to the hospital on Oct. 22, 1945. Five days before his admission, the patient vomited and had continued to vomit since. There were no bowel movements for two days, and abdominal pain was evident for two days. Pain came in spasms which increased in severity.

Results of physical examination were essentially normal, except that the bowel sounds seemed hyperactive at intervals, coming in gushes. The impression on the patient's admission was that he had intestinal obstruction.

Later in the day, physical examination revealed rigidity of the neck, stiffness of the back, but the abdomen was normal. A spinal puncture was done. The cell count and protein level were elevated.

Eyeground examination revealed choroiditis, juxtapapillary on the right. The Mantoux reaction (1:100,000) was negative. Two days later, the patient was fairly alert but had bilateral internal strabismus, more evident on the right. The pupils were equal and responded to light. There was nuchal rigidity; a suggestive Babinski reflex was elicited bilaterally, but no Kernig sign. Meningitis was now considered. Lumbar puncture was repeated, and the glucose level was 26 mg. Streptomycin, 200,000 units every three hours, was started.

On October 27, the patient did not look as alert as he had previously. He was irritable, had nuchal rigidity and showed the Brudzinski sign, with questionable presence of the Kernig. Pupils were equal but did not react well to light. Heart, lungs and abdomen were normal. The impression at this time was that he had tuberculous meningitis. On October 28, the patient had a convulsion. The entire body was involved. On October 29, the Mantoux test (1:10,000) showed a positive reaction.

The patient had a low grade fever and remained irritable.

He improved until April 8, 1948, when he had a convulsion limited to the left side. This was associated with horizontal nystagmus with the slow component to the left. He also had incomplete bilateral optic atrophy. He was walking and talking previously, but with this episode he became drowsy and irritable when aroused. He died on May 9, after seven months of hospitalization.

On autopsy the brain and meninges showed tuberculous meningoencephalitis. There was a thick exudate over the entire base of the brain. Numerous tubercles were observed over the surface of the brain. On opening the brain, a large granulomatous tuberculoma was seen in the right hemisphere. The lungs were edematous and the hilar nodes were enlarged and caseous.

CASE 5.—J. G., an 8 year old Mexican boy, was admitted to the hospital on Jan. 9, 1948, with a three week history of anorexia, slight cough, vague abdominal pain and fever. The mother had had tuberculosis during the preceding year. One year before, a sibling died of a similar condition. A survey of the family showed no evidence of tuberculosis in the other members.

On his admission, the child was afebrile but had moderate nuchal rigidity and was irritable. Examination of the chest revealed decreased breath sounds over the upper third, anteriorly and posteriorly. There was a lag over the entire right side of the chest. Nuchal rigidity and the Brudzinski and Kernig signs were present. A diagnosis of tuberculous meningitis and pneumonitis was made.

The appearance on roentgenologic examination of the chest was consistent with the physical findings. Streptomycin, 200,000 units every three hours, intramuscularly, was given for ninety days.

About ten days after his admission, his abdomen became full, and right hemiplegia and paralysis of the right facial nerve developed. Six weeks after his admission the child showed herpes zoster along the eighth rib on the left side.

Two months after his admission, the facial paralysis and hemiplegia began to improve. Nuchal rigidity disappeared. Three months after his admission, however, he took a turn for the worse. The two lower extremities became spastic. He had bilateral strabismus, internal on the right and external on the left. The temperature rose to about 103 F. rectally. He died on April 27, seventeen weeks after his admission.

On autopsy the brain was edematous and congested. There were numerous small tubercles over the entire surface and a thick exudate over the brain stem. The appearance of the lungs was typical of miliary tuberculosis with thickening and effusion in the right pleural region. The hilar and mesenteric lymph nodes were enlarged and caseous. Loops of bowel were matted together by an exudate and numerous fibrous adhesions. A miliary spread of tuberculosis was evident in the liver, spleen, kidney and ileum.

SUMMARY

Nineteen patients with tuberculous meningitis were treated with streptomycin. The youngest patient was 6 months of age and the oldest, 13 years. Ten were boys and 9, girls; 15 were Negro, 3 Mexican and 1 white. Streptomycin, 200,000 units intramuscularly every three hours, was administered and treatment continued for ninety days.

Of the 19 children, 11 died and 8 are still alive. One is alive fifteen months after the onset of the disease, 1 thirteen months, 1 twelve months, 1 eleven and one-half months, 1 eight months, 1 five months and 2 three months. Of the 11 who died, 7 lived from seven weeks to seven months after the onset of the disease. Considering the fact that before the streptomycin era no patient lived beyond seven weeks, I am certain that streptomycin was responsible for prolonging these lives.

I found intrathecal administration of streptomycin unnecessary in the treatment of tuberculous meningitis.

Encephalitic symptoms were frequent and at times overwhelmed the meningitic symptoms.

The most important complications observed were paralysis, optic atrophy and deafness. In one patient the deafness was due to the meningitis proper. In the other, it may have been due to the streptomycin.

In a few instances, the red blood cell count was increased beyond 6,000,000. In several patients, there was eosinophilia of varying degree during the third month of treatment.

TABLE 12.—*Summary of Data on Patients with Tuberculous Meningitis Who Received Streptomycin Therapy*

Total Patients Treated		19
Negro	15	
Mexican	3	
White	1	
Boys	10	
Girls	9	
Improved	8	
Died	11	
Lived more than 7 weeks	7	
Lived less than 7 weeks	4	
Relapses	7	

The cerebrospinal fluid was xanthochromic in 5 children. The cell count showed a rise after the beginning of the streptomycin treatment and then a recession. The same was true of the protein level. The values for glucose were most unusual in that they rose quickly, then fell again and gradually returned to normal.

The streptomycin level in the blood, taken three hours after administration of streptomycin, varied between 0 and 160 units. In the cerebrospinal fluid, the level varied from 0 to 320, with an average of 5 to 20 units. In a few instances the streptomycin level in the cerebrospinal fluid was higher than in the blood.

CONCLUSION

Streptomycin is of great value in the treatment of tuberculous meningitis. It is, however, too early to state whether the patients treated with streptomycin are completely cured. In fact, one must not consider any patient with tuberculous meningitis completely recovered, as relapses may occur at any time and may result fatally.

However, in consideration of the almost 100 per cent mortality associated with tuberculous meningitis under all previous methods of treatment, all patients with tuberculous meningitis should be treated with streptomycin.

30 North Michigan Boulevard.

ABSTRACT OF DISCUSSION

DR. W. W. BRIANT JR., Pittsburgh: Reports from a recent meeting in New York reveal that the mortality from miliary tuberculosis in the series described was 80 per cent in children. The 20 per cent salvage is credited to streptomycin. Failure of streptomycin to cure tuberculous meningitis may be due to the drug's failure to control miliary tuberculosis, the source of the hematogenous infection in many cases. It might be argued that the encephalitic phase of tuberculous meningitis results in irreversible changes, but it is apparent that the permanence of the cure of the meningitis or meningoencephalitis depends in many cases on the rapidity of control of the original site of the hematogenous infection. It is small comfort to the clinician to see improvement in one area while the feeder area is controlled poorly, if at all.

A second impression gleaned from delving into the subject is that the frequency of neurologic complications is considerably greater in the series in which streptomycin is used intrathecally, without any corresponding increase in rate of survival to justify intrathecal administration.

Is the streptomycin dosage of 10 to 20 mg. per kilogram of body weight per twenty-four hours an adequate one, and, if so, would the use of more than 20 mg. per kilogram account for some of the toxic symptoms reported? It is questionable whether the dose per kilogram per twenty-four hours is as important as the duration of treatment in any attempt to evaluate the toxicity of the drug. This is not a criticism of streptomycin as employed to date. The philosophy still prevails that in the absence of definite contraindications, the dose of streptomycin, as of morphine, should be enough to accomplish the desired result.

Dr. Maud L. Menten, the pathologist to the Children's Hospital, Pittsburgh, reports that since the advent of streptomycin therapy, for the first time in her experience, sections of brain from patients with tuberculous meningitis show a loosening of the perivascular mantle of monocytes phagocytosing tubercle bacilli and a replacement of these monocytes by lymphocytes. In these areas are evidences of beginning fibrosis. This picture is characteristic of healing tuberculosis elsewhere. Some sections of brain also contained giant cells, which are rare in untreated tuberculous meningitis.

DR. G. J. LEVY, Memphis, Tenn.: This carefully prepared presentation by Dr. Levinson gives us an insight into what the future holds toward the conquest of tuberculous meningitis. The term "cure" has rightfully been omitted, for who among us at present has the authority or proof that relapses are not likely to occur? I am in accord with regard to the spectacular responses of streptomycin therapy in tuberculosis. Patients apparently moribund in two or three days become quite alert. Their temperature response, too, is dramatic in some cases.

Dr. Corper and Dr. Cohn in a recent article in *The Journal of the American Medical Association* on "Various Phases of the Use of Streptomycin in Tuberculosis" (**137**:357 [May 22] 1948) stressed that streptomycin cannot be considered a cure in man at present, since it does not destroy virulent tubercle bacilli nor does it completely retard their development in vivo. In their experimental virulent infection, streptomycin did retard tuberculosis but could not prevent a lethal issue of the infection. With these facts in mind, I still must agree with Dr. Levinson that streptomycin is of great value in the treatment of tuberculous meningitis and should be administered in every case.

Before the advent of streptomycin there was universal agreement that tuberculous meningitis carried 100 per cent mortality. In twenty-eight years, I have seen 3 patients who recovered, all treated symptomatically. This treatment consisted of frequent lumbar punctures and hypertonic glucose solution intravenously. Since 1947, my associates and I have treated 11 patients with the disease at the Isolation Department of the John Gaston Hospital, Memphis, Tenn. All patients were treated intrathecally with 50 mg. of streptomycin for a period of seven days and 125,000 units intramuscularly every three hours. In our series there were 5 boys and 6 girls. Five were Negro, 5 white, and 1 Mexican. Three of the 11 are still alive. Two of the patients died within twenty-four hours after admission.

One white boy 13 years of age is alive seventeen and one-half months after treatment and apparently in good health. He has gained 40 pounds (18.1 Kg.) in weight and is back in school. This patient received more than 200,000,000,000,000 units of streptomycin in treatment. One Negro boy 12 years of age is alive after two months. He had bilateral paralysis of the sixth nerve, but this has corrected itself to a large extent. One white infant girl 8 months of age is alive after three months. The patient has hydrocephalus and generalized spasticity.

DR. WILLIAM S. HOFFMAN, Chicago: Dr. Levinson is to be congratulated for his courageous management of these patients. So far as I know, this is the first report of successful treatment of tuberculous meningitis with streptomycin without resort to intrathecal injections. It had been assumed that, because streptomycin is normally absent from the spinal fluid after intramuscular or intravenous injections, such injections alone cannot be effective in the treatment of the meningitides and that they must be supplemented with intrathecal injections. The present report gives ample evidence, both from the therapeutic point of view and from the streptomycin assays, that in tuberculous meningitis streptomycin is able to penetrate satisfactorily into the cerobrospinal fluid, possibly because of the inflammatory changes in the

choroid plexus or in the meninges themselves. If the cerebrospinal fluid levels obtained under such circumstances are, as shown here, in the therapeutic range, then intrathecal injection, with its attendant potentialities for irritation and contamination and with its physical and psychologic assault on the patient, is unnecessary. My co-workers and I have had some similar success with the use of massive doses of penicillin intramuscularly in the treatment of meningococcic meningitis.

The apparent lack of correlation between blood and cerebrospinal fluid levels of streptomycin, I believe, is well explained by the relative speeds of removal of streptomycin. It appears that in the first hour or so after intramuscular injection, the blood levels are much higher than the spinal fluid levels, but in later hours streptomycin is rapidly disappearing from the blood, but only slowly from the spinal fluid. The spinal fluid levels are, therefore, higher at this time.

DR. ABRAHAM LEVINSON, Chicago: The two questions that call for an answer most frequently are: 1. When is the patient ready to be sent home? 2. What should be done in case of a relapse? I send my patients home after an observation period of at least a month following the completion of streptomycin therapy, if the patient is clinically well and the constituents of the cerebrospinal fluid are within normal limits. As to relapse, I give streptomycin again and continue the treatment for one month or longer, depending on the condition of the patient. Among the laboratory problems, Dr. Briant told us what happened to the brains of children who died of tuberculous meningitis. Before the streptomycin era, there occasionally was some spontaneous healing, even in patients who died of the disease. My associates and I are now studying the brains of patients who died in spite of streptomycin therapy, and we shall prepare a report at some future date.

The most significant fact to note is that patients need no longer die without treatment, for, thanks to streptomycin, physicians are not as helpless as they used to be. They realize that streptomycin is of great value in the treatment of tuberculous meningitis. It is still too early to state, however, whether streptomycin alone suffices or is more effective when used in combination with other therapeutic agents. We do know with certainty that we can hope for a prolongation of life, if not for complete recovery, in a disease that had been 100 per cent fatal.

TREATMENT OF INFANTILE CONGENITAL SYPHILIS

Results with Aqueous Penicillin Alone in Sixty Infants Followed for an Average of Two Years After Treatment

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AND
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IN THE treatment of the infant with congenital syphilis, general experience to date has proved penicillin the most satisfactory therapeutic agent. Long term follow-up reports, however, have been lacking, and it is for this reason that our study, based on results in 60 infants [1] followed over an average of two years, is presented. Previously, seemingly adequate antisyphilitic therapy in infancy has been followed by late relapses, the development of paresis or interstitial keratitis.

A previous report [2] discussed selection of cases, dosages, reactions, complications, deaths and immediate response to treatment. This report concerns itself more with long term observation of whether or not clinical or serologic relapses may take place in spite of initial clinical improvement and whether or not any other complications or sequelae of congenital syphilis may occur.

This study was made with the collaboration of John H. Stokes, M.D.; John W. Lentz, M.D.; Virgene S. Wammock, M.D.; O. M. Carrozzino, M.D., and Herman Beerman, M.D., and the technical assistance of Emily Stannard, Verna Mayer Stein, Jane Barbara Taylor, R.N., and Mildred F. Wills, R.N.

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From the Institute for the Study of Venereal Disease, University of Pennsylvania, with the United States Public Health Service cooperating, and the Penicillin-Syphilis Panel of the Hospital of the University. The Institute, the Departments of Pediatrics, Neurology, Ophthalmology and Dermatology and Syphilology; the Division of Venereal Disease Control, Philadelphia City Department of Public Health, and the Philadelphia General and Pennsylvania Hospitals and Children's Hospital are represented in the authorship.

1. The Philadelphia General Hospital supplied 22 of these infants; the Children's Hospital, 16; the Pennsylvania Hospital, 4; the Hospital of the University of Pennsylvania, 12. One each were from the St. Luke's and Children's Medical Center, Delaware County Hospital, St. Joseph's Hospital, Metropolitan Hospital, Frederick Douglass Memorial Hospital and Dr. J. Winfree.

2. Rose, E. K.; György, P., and Ingraham, N. R., Jr.: Penicillin in the Treatment of the Syphilitic Infant: Progress Report, J. Pediat. **29**:567, 1946.

RESULTS OF TREATMENT

Table 1 shows an analysis of the age at onset of therapy in the 55 patients treated with intramuscularly administered penicillin alone. The procedure with 5 other patients who received penicillin by mouth will be mentioned subsequently. No previous antisyphilitic treatment had been given except to 2 of the 60 children. In both instances, the amount of treatment was considered too small to influence the results in our study. No patient was accepted for treatment without a positive reaction to a serologic test for syphilis and, in addition, either dermatologic or

TABLE 1.—*Age at Onset of Treatment and Mortality*

Age, Months	Total No. of Infants Treated	Number Dead	Per Cent Mortality
Under 3	33	6	18.2
From 3 to 6	10	1	10.0
From 6 to 12	5	0	0
From 12 to 24	5	0	0
Over 24	2	0	0
Total	55	7	12.7

TABLE 2.—*Various Dosages Used*

Number of Patients	Dosage	
	Units per Pound of Body Weight	Units per Kilogram of Body Weight
29	Up to 40,000	< 88,000
14	40,000 to 80,000	88,000 to 176,000
11	80,000 to 200,000	176,000 to 440,000
1	Over 200,000	>440,000
Total 55		

roentgenographic evidence of the disease. Aqueous penicillin of known brand and lot number was given intramuscularly every three hours in periods varying from 7 to 16 days. Dosage was varied as experience grew. During the earlier part of the study, between 11,000 and 20,000 units per pound (24,200 to 44,000 units per kilogram) of body weight for the total dosage was employed. This was gradually increased to 200,000 units per pound (440,000 units per kilogram) of body weight, given over a period of 15 days, in equal doses, every three hours.

In none of the 7 deaths, discussed fully in our previous report,[2] was it felt that the penicillin therapy was related to the outcome. Two of the 7 infants died later at home of other causes after definite clinical improvement in their syphilis. The other 5 were all small, debilitated infants who probably would never have lived to be included in the study

had supportive treatment without penicillin been inaugurated first. In every infant treatment was begun immediately as soon as the diagnostic criteria were satisfied.

Table 2 shows the number of patients receiving the various dosages.

Follow-up studies on our treated patients included regular visits, at which time were noted serologic titers, physical examination and roentgenologic examination of the bones when previous abnormalities existed. Yearly examination of cerebrospinal fluid was planned. The

TABLE 3.—*Duration of Observation*

Number of Patients	Period of Observation, Yr.
12	Under 1
19	Between 1 and 2
11	Between 2 and 3
10	Over 3
1	Lost to follow-up study
7	Died
Total 60	

TABLE 4.—*Outcome Analyzed as to Age at Which Treatment Was Started in Fifty-Five Patients Followed an Average of Two Years*

Age at Onset of Therapy, Months	Total Number Treated	Number Dead	Number Living	Number Apparently Cured (Sero-negative Reaction)	Number Clinically Well: Sero-Positive Reaction or Declining Titer	No Change in Titer
Under 3	33	6	27	27	0	0
3 to 6	10	1	9	5*	3	1 lost
6 to 12	5	0	5	2	3	0
12 to 24	5	0	5	2	1	2
Over 24	2	0	2	0	1	1
Total	55	7	48	36	8	4

* One patient was retreated at 177 days because of a positive result on dark field examination. His reaction was seronegative from 289 to 1,432 days.

average length of the period of observation was 2 years on 52 of the 53 surviving patients—only 1 patient having been lost to follow-up study. The shortest follow-up periods were 59, 85, 92 and 106 days, and the longest, 1,339, 1,343, 1,362 and 1,432 days. Table 3 shows the duration of observation. Our plan was to continue yearly examinations of these children, with especial attention to their serologic titers, growth, changes in spinal fluid and possible complications, such as interstitial keratitis or juvenile paresis in those who were seroresistant.

Response to therapy is shown in table 4 with relation to the age at onset of treatment. In the group under 3 months of age were 33 infants,

of whom 27 lived and were followed. All were clinically well; the reactions of all 27 remained seronegative. None showed relapse. Their serologic titers fell uniformly and, on an average, reactions of all patients were seronegative and remained so from 198 days on. This figure might have been less had regular monthly titers been obtainable on all patients, but this difficulty in a regular follow-up program is common to all such studies. The rate of the fall in titer seemed to bear no relation to the amount of penicillin used, as the total dosages in this group varied from 11,000 to 180,000 units per pound (24,200 to 396,000 units per kilogram) of body weight.

In the next older group of 9 infants (between 3 and 6 months of age at onset of treatment), reactions of 3 became and remained seronegative at 135, 350 and 365 days. The reaction of another was seropositive at 83 days, but, when the infant was next seen at 689 days, the reaction had become seronegative. Another, treated at 16 weeks with 18,000 units per pound (36,000 units per kilogram), was retreated at 177 days when he showed relapse, a positive result on dark field examination. His reaction became seronegative at 289 days and remained consistently so through 1,432 days. Three others became clinically well; the reactions of 2 of these were still seropositive, since they were treated only 85 days previous to this report, and the third infant's test had a doubtful reaction at 414 days. One was lost to follow-up study. Here again the clinical response seemed unrelated to the amount of penicillin used, the dosages ranging from 18,000 to 180,000 units per pound (39,600 to 396,000 units per kilogram) of body weight.

Of the 5 infants between 6 and 12 months of age, all responded well clinically, but 3 still showed declining titers—4 Kline units at 106 and 208 days respectively in 2. The third showed a doubtful reaction at 612 days. Interestingly enough, this last patient, S. M. (case 4) received the largest dosage of any of our patients, 455,000 units per pound (1,010,000 per kilogram). This dosage was chosen to determine what, if any, effect it might have on the severe and extensive osseous lesions. This phase of our study will be discussed in a subsequent report.

Of the 5 infants between 12 and 24 months of age, all became clinically well but varied in serologic response. The reactions of only 2 were seronegative, one at 314 days and the other at 955 days, but only after treatment at 386 days. One infant showed a declining titer at 59 days, when he was lost to follow-up study. Another showed no change in titer, yet he received his treatment of 200,000 units per pound (440,000 units per kilogram) of body weight only 92 days previous to this report. A third infant, after initially receiving 40,000 units per pound (88,000 units per kilogram) and continuing to show 256 Kline serologic units, was retreated at 520 days.

Of the 2 infants over 2 years of age, both were well, but their reactions were still seropositive. One showed a declining titer 538 days after receiving 68,000 units of penicillin per pound (149,600 units per kilogram) of body weight. The other was retreated 215 days after an

TABLE 5.—*Data on Infants Treated with Penicillin Who Became Clinically Well But Still Had Seropositive Reactions*

Case Number	Age at Onset of Treatment	Dosage of Penicillin		Comment
		Units per Pound of Body Weight	Units per Kilogram of Body Weight	
1	3½ mo.	180,000	396,000	Titer still 256 Kline units at 85 days
2	5 mo.	70,000	154,000	Declining titer, doubtful reaction at 464 days
3	5½ mo.	180,000	396,000	Titer still 256 Kline units at 85 days
4	6 mo.	455,000	1,010,000	Declining titer, doubtful reaction at 612 days
5	7 mo.	180,000	396,000	Declining titer, 4 Kline units, at 106 days
6	9 mo.	20,000	44,000	Declining titer, 4 Kline units, at 208 days
7	16 mo.	40,000	88,000	Clinically well, but no change in results of serologic tests (256 Kline units); retreated at 520 days
8	18 mo.	200,000	440,000	Clinically well, but no change in results of serologic tests (256 Kline units); retreated at 92 days
9	30 mo.	20,000	44,000	Clinically well, titer still 128 Kline units at 215 days; retreated; reaction still positive (North Carolina) at 474 days
10	2 yr.	70,000	154,000	Declining titer, 64 Kline units, at 538 days

TABLE 6.—*Results in Five Infants Treated with Penicillin by Mouth*

Case Number	Age at Onset of Therapy	Dosage in Million Units	Comment
11	20 wk.	1.5	Titer still 256 units at 385 days when retreated with 1,200,000; negative reaction at 821 days
12	18 wk.	1.5	Negative reaction at 129 days and still at 742 days
13	7½ wk.	1.3	Negative reaction at 318 days through 885 days
14	12 wk.	2.5	Negative reaction at 298 days through 500 days
15	2½ yr.	2.25	Titer still 64 units at 466 days; retreated; titer still 64 units at 877 days; retreated second time

initial dosage of 20,000 units per pound (44,000 units per kilogram) because her serum still showed 128 Kline units. She then moved to North Carolina, where a positive reaction to a serologic test was reported at 474 days (table 5).

In the 16 patients on whom repeated examinations of the cerebrospinal fluid were obtained, no abnormalities were noted. These patients all had satisfactory serologic and clinical responses.

An attempt was made to study the effects of penicillin given by mouth. Five infants were treated (table 6). A buffered preparation of calcium penicillin was used in dosages varying from 1,500,000 to 2,500,000 units. Tablets of 12,500 units were given every three hours. No untoward effect was noted, except looseness of stool occasionally in the younger infants. Results are noted in table 6. Here again it is demonstrated that the best results were obtained in the younger infants.

COMMENT

It would seem, then, that one might expect good clinical response in congenital infantile syphilis to treatment with penicillin given intramuscularly within a fairly wide range of dosage. Perhaps the most important factor favoring good response is the age of the patient at the onset of treatment—the younger the patient the better the outcome. The fact that there was not a single failure in therapy among the surviving infants, treated under the age of 3 months, is particularly noteworthy. This is in accord with observations on the experimental animal and on the adult that syphilis is extremely amenable to treatment in its initial stages. Although clinical improvement is an almost uniform response in infants of all ages, the fall in serologic titer becomes increasingly less prompt and persistent in the older infants. Penicillin was thought to have no relation to the cause of death in our infants. This has been previously reported.[2]

The long term follow-up study here presented seems to bear out the previous impression of the remarkable superiority of treatment with penicillin in congenital infantile syphilis over all other previous therapeutic schemes and recommendations, not only with regard to the safety and relatively short duration of therapy, but also in respect to the lack of clinical and serologic relapses. There was no indication that various penicillin preparations (amorphous or crystalline) behaved differently.

SUMMARY

Sixty infants with congenital syphilis were treated with penicillin and followed for periods up to three years.

Of the 53 living patients, only 1 was lost to follow-up study.

Of the 48 living patients treated with penicillin intramuscularly, 37 were apparently cured (clinically well and with seronegative reactions); 7 were clinically well but still showed seropositive reactions, though the titer was declining; 3 were well but revealed no change in titer. Two of the latter were recently retreated.

Five patients were treated with penicillin by mouth, 4 infants responding satisfactorily. One older child 2⅓ years of age responded well, but his reaction remained seropositive after retreatment.

The age of the patient at the onset of treatment, rather than the dosage, type or means of administration of penicillin, seemed to be the chief factor in determining satisfactory response; cures approached 100 per cent when treatment was commenced before the third month of life. The amounts of penicillin administered intramuscularly varied from 20,000 to 200,000 units per pound (44,000 to 440,000 per kilogram) of body weight. Even in this relatively small group of 53 patients studied over prolonged periods up to three years, the absence of serologic relapse, except in the case cited, should be emphasized.

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COMPARISON OF VITAMIN B_1 LEVELS IN MOTHERS AND THEIR NEWBORN INFANTS

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THE PLASMA ascorbic acid concentrations of the newborn infant and of cord blood were shown to be considerably higher than that of the maternal blood.[1] Similar studies with the intradermal test[2] for vitamin C gave results which are in agreement with these reports of maternal-neonatal ascorbic acid levels of plasma. These studies suggested the important concept that the fetus takes its supply of vitamin C whether or not the mother has a sufficient amount.

The question whether other vitamins act in a similar fashion should be investigated. It seemed worth while, first, to study the relationships of thiamine in the mother and her newborn infant. In previous studies with determinations of thiamine levels in the blood of children, we observed that values for thiamine were more stable than those for ascorbic acid. In children there was a normal range of 4.8 to 12.3 micrograms per hundred cubic centimeters, with a mean of 7.8 $\pm$ 1.3 micrograms.[3]

From the Frederick S. Wheeler Research Laboratory and the Pediatric Department of the New York Medical College, Flower and Fifth Avenue Hospitals.

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The vitamin B_1 content of the blood did not fall until there was a severe clinical deficiency of thiamine.[4]

PLAN OF STUDY

We planned to compare the vitamin B_1 levels of maternal and cord blood and the blood of newborn infants. The cases were unselected and taken as consecutive deliveries. These newborn infants were breast fed. The blood from the infant was taken on the fifth day of life. An accurate thiochrome method[5] which determines the total vitamin B_1 content of the blood was used. This method had been employed in all our previous studies.

RESULTS

The results are presented in the table. There were 48 determinations on maternal and cord blood, and 35 determinations on the blood of the 5 day old infants. In the mothers, the lowest blood thiamine value was 2.45 micrograms and the highest, 10.19. The mean was 6.97 micrograms. The lowest cord blood level was 4.90, and the highest was 20.59 micrograms. The mean was 11.63 micrograms. In the 5 day old infants, the lowest blood thiamine value was 2.08 micrograms and the highest was 15.80. The mean in these newborn infants was 9.57 micrograms.

The thiamine level in the cord blood was higher than in the maternal blood in 47 of the 48 individual cases. The vitamin B_1 of the 5 day old newborn infants was also higher than that of the mothers, except in 1 instance. In 25 of the infants, the thiamine value was lower than that of the cord blood.

COMMENT

The mean thiamine value for cord blood was almost twice that for the maternal blood. This high cord blood level falls by the time the newborn infant reaches five days of life. These results with vitamin B_1 are in substantial agreement with previous reports on maternal-neonatal ascorbic acid relationships.[6]

The recent studies of Hamil and his associates[7] on the urinary excretion of certain of the vitamins of the B complex in the newborn are outstanding in explaining this maternal-neonatal relationship. The

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Comparison of Thiamine Levels in the Maternal and Cord Blood and the Blood of Newborn Infants

		Infants, 5 Days of Age			Mother		Cord Blood,
Case No.	Race	Sex	Weight at Birth, Kg.	Vitamin B_1, Micrograms/ 100 Cc.	Age, Yr.	Vitamin B_1, Micrograms/ 100 Cc.	Vitamin B_1, Micrograms/ 100 Cc.
1	W	F	2.91	2.08	26	2.45	17.31
2	N	M	3.52	8.01	24	4.80	10.09
3	N	M	2.13	9.44	18	4.90	6.27
4	W	F	3.23	4.26	36	5.39	11.06
5	W	F	2.99	....	19	5.49	10.58
6	N	F	4.14	10.27	19	5.69	10.10
7	W	M	3.27	9.80	20	5.79	11.66
8	W	F	1.36	....	19	5.87	11.47
9	N	M	3.74	7.62	23	5.88	9.62
10	W	F	2.44	10.78	22	5.98	12.84
11	N	M	2.27	9.51	21	5.98	11.66
12	W	M	3.69	7.94	28	6.08	9.12
13	N	M	3.86	14.80	27	6.08	4.90
14	N	M	2.80	7.95	24	6.08	10.29
15	W	M	3.29	....	19	6.18	11.57
16	W	M	3.42	9.48	32	6.27	20.40
17	W	F	3.30	7.72	25	6.37	7.75
18	N	M	3.46	10.79	24	6.37	10.69
19	N	F	2.74	....	20	6.37	10.88
20	W	F	2.90	11.56	27	6.47	8.82
21	W	F	2.78	10.79	22	6.57	18.07
22	N	M	2.49	11.27	18	6.67	8.92
23	N	M	3.37	9.43	21	6.76	12.06
24	N	M	3.37	7.26	30	6.96	9.71
25	N	M	2.40	15.80	23	6.96	13.03
26	W	M	3.63	7.16	20	7.06	12.16
27	N	M	2.82	....	26	7.06	9.21
28	N	M	4.14	....	26	7.16	13.92
29	N	F	3.57	11.95	40	7.20	11.30
30	W	F	3.73	10.29	27	7.34	11.48
31	N	F	2.66	....	19	7.35	10.29
32	N	M	3.18	10.91	19	7.35	10.59
33	W	F	3.46	11.95	34	7.40	11.30
34	N	F	2.98	9.31	26	7.55	11.27
35	N	F	3.90	....	18	7.75	10.79
36	W	M	3.60	7.84	27	7.83	8.43
37	W	F	4.19	....	39	8.23	13.72
38	W	M	3.69	....	36	8.24	8.73
39	W	M	2.64	....	21	8.24	9.90
40	W	F	3.19	....	18	8.24	16.34
41	W	M	3.02	8.04	19	8.33	10.78
42	W	F	2.89	9.97	24	8.39	11.35
43	W	M	3.66	9.61	21	8.53	11.47
44	W	F	2.99	....	19	8.73	20.59
45	W	M	3.30	10.59	35	8.92	15.39
46	N	F	3.60	11.54	32	9.31	13.04
47	N	F	2.79	9.31	31	9.41	15.87
48	N	F	3.15	9.90	22	10.19	12.24

urinary excretion of these vitamins was high during the first few days of life and fell off greatly by the seventh day. Hamil and his associates suggested an interesting thought, that, in the relatively anoxic intrauterine state, these vitamins are needed in excess and are stored in the fetal tissues. The transition to the extrauterine environment results in the excretion of the now excessively large amounts of these vitamins during the first few days of newborn life.

The need in the relatively anoxic intrauterine state would not explain the actual mechanism by which the levels of vitamins B and C are built up and the substances stored in the fetal tissues. Many theories have been suggested to explain this mechanism. None are completely satisfactory. Lund and Kimble[1c] presented good evidence that the placenta acts selectively to retain the vitamin C in the fetus. The entire answer cannot be given and may await working out of such phases as nervous-humoral-vitamin relationships and controls.

At present, the several ideas can be combined into a reasonable hypothesis. It is known that thiamine and its co-enzyme take part in the oxidative enzyme systems which act in cellular respiration. During fetal life, due to the relatively low oxygen tension, larger amounts of the vitamin are required. This excess of the vitamin is taken from the mother, perhaps by means of the placenta acting selectively to retain the necessary quantities. After the fetus is delivered, there is a relatively high oxygen tension. The amount of the vitamin in the newborn tissues is now above the level necessary for good function. This excess is then excreted. The ultimate controls of the entire relationship require further investigation.

At any rate, it is clear that the fetus will take its supply of these vitamins from the mother, whether or not she has a sufficient amount. Therefore, the pregnant mother should be assured of an adequate supply of these vitamins in the diet or by supplementation.

SUMMARY

1. A comparison of the vitamin B_1 levels in mothers and their newborn infants was made.

2. In the mothers, the mean thiamine level in the blood was 6.97 micrograms per hundred cubic centimeters. The mean thiamine level in cord blood was 11.63 micrograms. In the 5 day old newborn infants, it was 9.57 micrograms.

3. These results of the determination of thiamine in the blood are similar to previous reports on maternal-neonatal ascorbic acid relationships.

4. The significance of these observations is discussed.

Dr. Andrew C. Brunhofer and Dr. Donald P. Beirne cooperated in this study.

Fifth Avenue at One Hundred and Fifth Street.

URINARY GLYCOCYAMINE, CREATINE AND CREATININE

I. Their Excretion by Normal Infants and Children

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AND
ROY W. PINELLI, M.D.
SAN FRANCISCO

THE purpose of this report is to record the quantitative urinary excretion of glycocyamine, creatine and total creatinine by a group of normal infants and children.

The metabolism of creatinine is initiated in the renal epithelium where glycocyamine, or guanidoacetic acid, is synthesized from the two amino acids, glycine and arginine.[1] Dubnoff and Borsook [2] showed that in the normal adult 5 per cent of the glycocyamine is excreted in the urine and the remainder is transported to the liver, where it becomes the accepter of a methyl radical in the transmethylation process. Methionine and the system of choline and homocystine act as the methyl donors to form methylglycocyamine, or creatine. Most of the creatine is then converted to its anhydride, creatinine, and excreted as such in the urine, along with varying amounts of unconverted creatine.

As far as is known, this is the only course followed by glycocyamine in the metabolism of creatinine, and it is graphically presented in the figure. This metabolic chain of events lends itself well to quantitative and radioactive isotopic analysis.

Hoberman [3] and his associates concluded that the total creatinine lost daily in the urine is replaced by an equimolar amount of creatine, synthesized from equimolar amounts of glycocyamine from the kidney, and methyl groups derived from the methyl donors in the liver. They further showed that small defects in the transmethylation process in the liver are reflected by a decided increase in the urinary output of glycocyamine.

From the Children's Department, St. Mary's Hospital.

1. Borsook, H., and Dubnoff, J.: Formation of Glycocyamine in Animal Tissues, J. Biol. Chem. **138**:389 (March) 1941.

2. (*a*) Dubnoff, J., and Borsook, H.: A Micromethod for Determination of Glycocyamine in Biological Fluids and Tissue Extracts, J. Biol. Chem. **138**:381 (March) 1941. (*b*) Borsook, H., and Dubnoff, J.: Methylation of Guanidoacetic Acid by Homocystine Plus Choline with Rat Liver Slices, ibid. **160**:635 (Oct.) 1945.

3. Hoberman, H. D.; Lloyd, C. W., and Williams, R. H.: The Role of the Liver in Guanidoacetic Acid Metabolism in Man, Science **104**:619 (Dec. 27) 1946.

The studies of this metabolic process with radioactive isotopes showed that when methionine containing deuterium in its methyl group, deuteriomethionine, was fed animals on a diet otherwise free from choline and methionine, creatinine containing deuterium was recovered from the urine.[4]

PROCEDURE

Seventeen infants and children, ranging from a premature baby of 2.3 Kg. to a child of 36.9 Kg., were used in the experiment. With one exception, they were

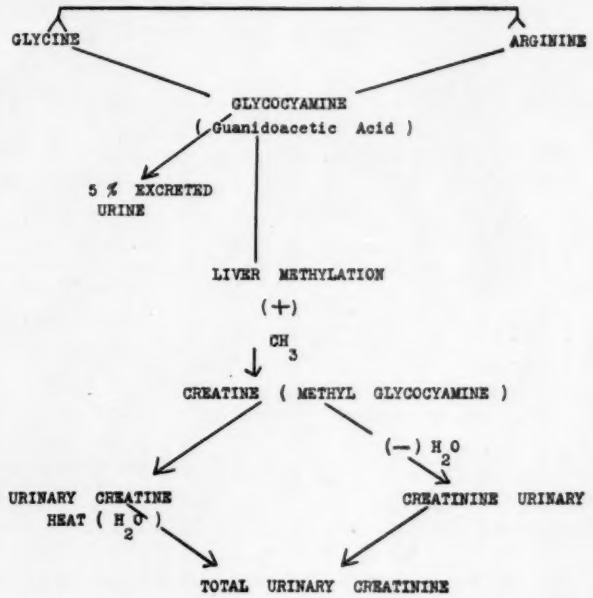

Diagram showing renal synthesis of glycine and arginine to creatinine by the transmethylation process.

normal subjects. The exception was a dehydrated infant awaiting operation for pyloric stenosis, whose urinary output was decidedly reduced. The distribution as to weight was as shown in the tabulation.

Weight, Gm.	Number of Patients
2,000 to 2,800	4
2,801 to 5,000	6
5,001 to 36,900	7

The urine was most accurately collected over a twenty-four hour period. This procedure was carried out in incontinent children with specially constructed plastic genital cuffs, which carried the urine into a length of rubber tubing, and thence into iced collecting flasks.

4. Kleiner, I. S.: Human Biochemistry, St. Louis, C. V. Mosby Company, 1945. du Vigneaud, V., and others: Utilization of the Methyl Group of Methionine in the Biological Synthesis of Choline and Creatine, J. Biol. Chem. **140**:625 (Aug.) 1941.

TABLE 1.—*Data Obtained on Study of Urinary Excretion of Glycocyamine, Creatine and Creatinine in Infants and Children*

Patient	C. A.	W. I.	R. R.	A. R.	K. Ea.	S. M.	M. O.	P. E.	G. R.	B. A.	S. T.	M. I.	C. A.	K. Eb.	S. H.	G. O.	M. A.
Age	5 days	2 days	21 days	6 days	6 days	15 days	30 days	18 days	6 wk.	10 wk.	2½ yr.	4½ yr.	7 yr.	6 yr.	7 yr.	9 yr.	13 yr.
Body weight, Kg	2.3	2.3	2.3	2.8	3.2	3.2	3.82	3.3	3.6	5.0	13.3	18.0	22.0	22.5	24.7	28.8	36.9
24 hour urine, cc	160	91	70	240	330	400	320	250	375	320	205	320	600	455	820	522	1,035
Glycocyamine																	
Mg./100 cc. urine	0.99	2.2	3.1	1.0	0.94	0.99	2.8	0.97	0.99	1.2	8.3	8.9	1.6	5.4	5.2	5.0	5.0
24 hour output, mg.	1.58	1.98	2.17	2.4	3.1	3.96	8.96	2.42	3.71	3.84	17.0	28.5	33.6	24.6	42.6	26.1	51.7
Mg./Kg. body weight	0.68	0.86	0.94	0.85	0.96	1.23	2.34	0.73	1.03	0.76	1.28	1.58	1.5	1.0	1.7	0.9	1.4
Creatinine																	
Total, mg./100 cc. urine	17.2	24.0	50.0	20.5	18.4	25.6	20.0	18.8	18.1	45.2	108.1	114.7	102.0	117.6	108.0	105.2	114.4
Preformed, mg./100 cc. urine	17.1	19.2	49.2	19.5	18.3	24.9	18.7	18.4	17.0	44.9	90.0	112.1	78.0	90.0	89.1	77.2	95.0
Total 24 hour output, mg.	27.52	21.84	35.0	49.2	60.7	103.4	64.0	47.0	69.9	144.64	221.6	367.0	612.0	535.0	885.6	549.2	1,184.0
Preformed, 24 hour output, mg.	27.36	17.4	34.5	47.8	60.3	9.96	59.8	46.0	66.9	143.68	180.0	358.7	468.0	405.0	730.6	401.4	978.5
Creatinine coefficient $\left(\frac{\text{Mg. total creatinine/24 hr.}}{\text{Kg. of body weight}}\right)$	12.0	9.9	15.8	14.0	18.9	32.0	16.8	14.2	18.2	28.9	16.3	20.4	27.7	23.7	36.2	19.5	30.2
Creatinine																	
Mg./100 cc. urine	0.125	5.9	1.0	1.25	0.12	0.87	1.62	0.5	1.17	1.19	21.7	3.25	34.0	34.5	23.62	35.3	23.05
Mg./24 hr. urine output	0.2	5.3	0.7	2.9	0.39	3.50	5.18	1.25	5.49	3.8	43.4	10.4	204.0	145.2	198.68	201.5	237.4
Mg./Kg. of body weight	0.08	2.3	0.3	1.01	0.12	1.06	1.3	0.37	1.5	0.76	3.2	0.78	9.2	6.4	7.8	6.9	6.4
Glycocyamine-creatinine coefficient $\left(\frac{\text{Mg. glycocyamine in 24 hr. urine} \times 100}{\text{Total creatinine in 24 hr. urine}}\right)$	5.7	10.1	6.2	4.8	5.1	3.86	14.0	5.1	5.2	2.7	7.6	6.1	5.4	4.5	4.8	4.9	4.3
Comment	Premature	Premature	Pyloric stenosis	Premature													

The gylcocyamine was determined by the method of Dubnoff and Borsook,[2a] as modified by Sims.[5] The creatinine was determined by the method of Bonsues and Taussky.[6] The preformed creatinine was also determined, and, after heating, the total creatinine was measured.

The creatine was estimated by multiplying the difference between the preformed and total creatinine by the factor 1.25, as with the method used only 80 per cent of the creatine was converted to creatinine on heating.[7]

In order to correlate the relative amount of total creatinine, creatine and glycocyamine excreted by the various children in twenty-four hours, the amount of each substance was expressed in milligrams per kilogram of body weight. This figure was designated as the coefficient of the substance.

TABLE 2.—*Summary of the Ranges and Means of Values Obtained, According to Age Group*

	Age Group	No. of Observations	Range	Mean
Glycocyamine coefficient ($\frac{\text{Mg. glycocyamine in 24 hours}}{\text{Kg. of body weight}}$)	Premature infants	3	0.68 to 0.86	0.80
	Normal infants	7	0.73 to 1.23	1.14
	Children	7	0.90 to 1.70	1.33
Creatinine coefficient ($\frac{\text{Mg. total creatinine in 24 hours}}{\text{Kg. of body weight}}$)	Premature infants	3	9.9 to 14.0	11.9
	Normal infants	7	14.2 to 32.0	21.6
	Children	7	16.3 to 36.2	24.8
Creatine coefficient ($\frac{\text{Mg. creatine in 24 hours}}{\text{Kg. of body weight}}$)	Premature infants	3	0.08 to 1.0	0.45
	Normal infants	7	0.12 to 1.5	0.76
	Children	7	0.78 to 7.8	5.81
Glycocyamine-creatinine coefficient ($\frac{\text{Mg. glycocyamine} \times 100}{\text{Mg. total creatinine}}$)	Premature infants	3	4.84 to 10.1	6.86
	Normal infants	7	2.7 to 14.0	6.00
	Children	7	4.3 to 7.6	5.35

The relationship between the urinary glycocyamine and the total creatine was expressed as a coefficient obtained by multiplying the milligrams of glycocyamine excreted in twenty-four hours by 100, then dividing the product by the milligrams of total creatinine excreted over the same period.

RESULTS

For brevity all the observations on the experiment are shown in table 1. A summary of the range and mean values obtained for each age group is presented in table 2.

5. Sims, E. A.: Microdetermination of Glycocyamine and Arginine by Means of a Synthetic Ion Exchange for Chromithographic Separation, J. Biol. Chem. **158**:239 (March) 1945.

6. Bonsues, R. W., and Taussky, H.: On the Colorimetric Determination of Creatinine by the Jaffe Reaction, J. Biol. Chem. **158**:581 (May) 1945.

7. Albanese, A. A., and Wangerin, D. M.: The Creatine and Creatinine Excretion of Normal Adult Males, Science **100**:58 (July 21) 1944. Bonsues and Taussky.[6]

COMMENT

A study of these figures demonstrates certain facts and trends.

First, these premature infants were well endowed with the ability to synthesize glycocyamine. This ability originally was questioned by Dubnoff.[8]

Second, children of all age groups showed a trend to excrete the same amount of glycocyamine in twenty-four hours when compared on a basis of milligrams per kilogram of body weight.

Third, the impression was gained during the experiment that the excretion of glycocyamine may be relatively independent of the volume of urine voided in twenty-four hours. This premise was partially substantiated in the single observation on baby R. R., who was badly dehydrated and passed only 70 cc. of urine in twenty-four hours. This amount of urine, however, contained 2.17 mg. of glycocyamine, or 1.34 mg. per kilogram of body weight. This amount was well within the normal limits.

Fourth, the total creatinine excreted in twenty-four hours per kilogram of body weight showed a moderate trend to increase from prematurity to adolescence. This is in keeping with the dictum that the excretion of creatinine is dependent on the mass of active muscle of the individual person.[4] The body weight of young infants represents relatively more water than that of older persons.

Fifth, the extreme fluctuations in the excretion of creatine in all groups is difficult to explain. Apparently, some children form from creatine the anhydride creatinine more rapidly than do others. In such children, less creatine may appear in the urine.

Sixth, the relationship between the urinary glycocyamine and total creatinine, as represented by the glycocyamine-creatinine coefficient, demonstrated a trend to decrease somewhat in children of the older age group. The probable explanation is that the excretion of glycocyamine has a tendency to remain fairly constant per kilogram of body weight in persons of all age groups, while there is a trend for the total output of creatinine per kilogram of body weight to increase.

SUMMARY

The urinary excretion of glycocyamine, creatine and creatinine was determined in 17 subjects, ranging in age from prematurity to 13 years.

8. Dubnoff, J.: Personal communication to the authors.

Urinary glycocyamine tended to be excreted in equivalent amounts per kilogram of body weight in twenty-four hours for children of all age groups. The total excretion of glycocyamine in twenty-four hours may be only slightly affected by a considerable reduction in the daily amount of urine voided.

There is a trend for the total creatinine excreted in twenty-four hours per kilogram of body weight and the glycocyamine-creatinine coefficient to increase with advancing age.

The excretion of creatine was observed to be variable and unpredictable in children of all age groups.

Sister Theophane supervised the collection of urine from these children.

490 Post Street.

PINWORM INFECTION AND CHILDHOOD BEHAVIOR

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RIVERSIDE, R. I.

PINWORM infection has long been regarded with suspicion by physicians and parents alike so far as its effect on the behavior of children is concerned. The variety of symptoms described in standard texts [1] and current literature [2] emphasizes the lack of specificity of findings. The majority of recent studies, however, intimate that oxyuriasis is generally an innocuous infection, but that when a high degree of parasitism exists, in some children certain nonspecific gastrointestinal, perianal or behavioral complaints become manifest.

The observation of a high incidence of pinworm infection in children who were resident patients of a children's psychiatric hospital prompted an evaluation in this type of child of the symptoms ascribed to infection with that parasite. It was believed that this study might help clarify the significance of the multiplicity of complaints ascribed to

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From the Emma Pendleton Bradley Home.

1. Nelson, W. E.: Mitchell-Nelson Textbook of Pediatrics, ed. 4, Philadelphia, W. B. Saunders Company, 1946, pp. 516-517. Davison, W. C.: The Compleat Pediatrician, ed. 4, Durham, N. C., Duke University Press, 1943, vol. 2, p. 32. Gradwohl, R. B. H.: Clinical Laboratory Methods and Diagnosis: A Textbook on Laboratory Procedures with Their Interpretation, ed. 3, St. Louis, C. V. Mosby Company, 1943, p. 1752. Strong, R. P.: Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases, ed. 6, Philadelphia, The Blakiston Company, 1942, vol. 2, p. 1232. Craig, C. F., and Faust, E. C.: Clinical Parasitology, ed. 3, Philadelphia, Lea & Febiger, 1943, p. 283. Faust, E. C., in Brennemann, J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Company, Inc., 1941, vol. 2, chap. 35, p. 8.

2. (*a*) Weller, T. H., and Sorenson, C. W.: Enterobiasis: Its Incidence and Symptomatology in a Group of Five Hundred and Five Children, New England J. Med. **224**:143-146 (Jan. 23) 1941. (*b*) Miller, J. F., and Einhorn, N. H.: Oxyuriasis, Am. J. Dis. Child. **68**:376-381 (Dec.) 1944. (*c*) Sisk, W. N.: Effect of Phenothiazine on Intestinal Parasites, J. A. M. A. **122**:357-360 (June 5) 1943. (*d*) Jacobs, A. H.: Enterobiasis in Children, J. Pediat. **21**:497-503 (Oct.) 1942. (*e*) Cram, E. B.: Studies on Oxyuriasis: XXVIII. Summary and Conclusions, Am. J. Dis. Child. **65**:46-59 (Jan.) 1943. (*f*) Brady, F. J., and Wright, W.: XVIII. The Symptomatology of Oxyuriasis as Based on Physical Examinations and Case Histories of Two Hundred Patients, Am. J. M. Sc. **198**:367-372 (Sept.) 1939.

pinworm infection. Although the number of children would be small, this would be compensated for by the possibility of prolonged study by trained observers.

MATERIALS AND METHODS

The children were all inpatients for relatively long periods at the Emma Pendleton Bradley Home, a children's psychiatric hospital. The reasons for their referral were behavior problems, neuroses, psychoses and convulsive disorders. They were from 4 to 12 years of age and of both sexes. The social, economic and racial backgrounds were of all types. Forty children were studied (28 with pinworm infection and 12 controls).

On admission, detailed histories were taken, and in addition to other laboratory studies, each patient had three consecutive perianal swabs (NIH method, modified with scotch® tape [2e]) prior to the first defecation in the morning. Routine blood counts were repeated at monthly intervals, and when an eosinophilia of 5 per cent or more appeared, or when clinical symptoms were noted, perianal swabs were repeated on three consecutive occasions.

The children lived for several months in a controlled atmosphere under the scrutiny of resident pediatricians, psychiatric nurses and trained guides, hereafter designated as "observers."

The data obtained were of two types. The history taken on admission was used as a source to indicate parents' complaints. In addition, detailed sheets (listing symptoms and signs most commonly reported in oxyuriasis, plus a space for random remarks) were distributed to the observers without their knowing either that the study was related to pinworms or which of the patients were or were not infected. Then the procedure to be described was instituted. The completed reports were the basis for the study.

A two week period of observation was begun. At its completion, the appropriate sections of the sheets previously distributed were filled out, with the presence or absence of each item being checked for all patients.

The positive responses were considered representative of symptoms that could be attributed to pinworms in these patients. Then, half of the children with pinworms and half of those without were treated for a ten day period with enteric-coated® tablets of methylrosaniline chloride U. S. P. (gentian violet) in a daily dosage schedule of 10 mg. per year of age, given in three doses.

One week after completion of therapy, three consecutive perianal swabs were taken. In all treated children, they were found negative for pinworm. One week later, a week-long period of observation was begun on all patients. Then, after an interval of two weeks, a repeat period of observation was instituted for another week (making a total of two weeks of observation completed one month after treatment).

At this time, all patients were rated as "improved," "unimproved" or "worse," on the basis of the completed observation sheets. "Improved" meant that two or more symptoms had disappeared (without regard to intensity); "worse" meant that a new symptom had appeared and the old ones had remained, again without regard to the intensity of the old or new symptoms.

Because they had been most frequently mentioned in the literature, the following symptoms were looked for: restlessness, irritability, short attention span, emotional instability and hyperactivity. Other behavioral manifestations were insomnia, enuresis, encopresis, masturbation, nail biting, thumb sucking, nose picking and teeth gritting. Gastrointestinal symptoms included poor appetite, abdominal

pain, underweight, nausea and vomiting. Perianal symptoms comprised pruritus ani, anal cutaneous lesions and vaginitis, while symptoms classified as "general" were pallor, dark circles under the eyes and listlessness.

The conclusions were based on the following factors: (1) observations before treatment as compared with those in published reports; (2) "parents' complaints" on the child's admission compared with "observers' remarks" during the period before treatment; (3) the effect of anthelmintic treatment on the symptoms of the patients with and without pinworm infection, compared with the course of illness in similar children who received no specific treatment.

TABLE 1.—*Incidence of Symptoms Ascribed to Pinworm in Twenty-Eight Patients at Bradley Home Compared with Data in Other Recent Studies*

Source	Bradley Home	Weller and Sorenson [2a*]	Miller and Einhorn [2b] A	Miller and Einhorn [2b] B	Sisk [2c]	Jacobs [2d]
Total number of patients with pinworm	28	505	100	100	106	75
Symptoms	Incidence Given in Percentage					
Poor appetite	14	25	6	7	48	38
Abdominal pain	0	18	11	13	..	41
Underweight	32	3	6	1	..	41
Nausea and vomiting	0	0	..	..	+	..
Indigestion	0	0	..	..	11	..
Pruritus ani	0	17	..	..	26	..
Anal cutaneous lesions	0	3	13	59	..	27
Vaginitis	0	6	..	..	..	21
Nervousness †	44	21	3	3	16	49
Insomnia	3	..	1	7	78	38
Sleepwalking	0	..	..	..	13	..
Nightmares	0	..	..	..	33	..
Enuresis	35	12	1	4	5	17
Encopresis	0	..	1	4	..	..
Masturbation	14	3	..	..	..	..
Listlessness	7	..	2	1	..	..
Eyes, dark circles	7	..	..	..	+	..
Nail biting	0	..	..	..	..	..
Pallor	7	..	..	..	..	..
No symptoms	10	36	75	50	..	22

* Weller and Sorenson [2a] studied 505 children from medical wards and the outpatient department of Children's Hospital, Boston. Miller and Einhorn [2b] reported on 200 children in Panama; group A were hospital patients; group B were studied in the outpatient department. Sisk [2c] wrote on 106 children from an orphanage and school near Asheville, N. C., and Jacobs [2d] studied 75 children in the outpatient department of Stanford University Hospitals, San Francisco.

† Nervousness implies a composite of the following: restlessness, irritability, short span of attention, emotional instability and hyperactivity.

RESULTS OF STUDY

Comparison with Other Studies.—The information obtained before treatment from the records of 28 patients who showed the presence of pinworm was compared with that in published reports of children with pinworm infections (table 1). Perianal symptoms (pruritus ani, anal cutaneous lesions and vaginitis) were not noted in our patients, whereas the incidence of these symptoms in other studies was significantly high. Similarly, in the Bradley group, abdominal pain, nausea and vomiting were not observed. Poor appetite also was not an outstanding symptom, but the incidence of underweight was higher than in the majority of other studies.

"Parents' Complaints" Versus "Observers' Remarks."—The significance of symptoms attributed to pinworm infection was evaluated in

TABLE 2.—*Symptoms Possibly Referable to Pinworm Infection as Indicated by "Parents' Complaints" and "Observers' Remarks" Prior to Treatment*

Symptoms	Group A*		Group B		Group C	
	Parents' Complaints	Observers' Remarks	Parents' Complaints	Observers' Remarks	Parents' Complaints	Observers' Remarks
	Incidence Given in Percentage					
Poor appetite	20	20	0	7	25	8
Abdominal pain	46	0	15	0	0	0
Underweight	6	40	23	23	16	25
Nausea and vomiting	6	0	0	0	0	0
Vaginitis	0	0	0	0	0	8
Restlessness	33	40	15	63	8	33
Irritability	13	40	0	15	16	50
Short attention span	33	56	15	53	8	53
Emotionally unstable	20	36	53	61	60	40
Hyperactivity	13	40	0	38	16	16
Insomnia	33	6	23	0	16	8
Enuresis	6	46	7	23	40	16
Encopresis	0	0	15	0	0	8
Masturbation	0	13	0	15	0	8
Listlessness	6	6	15	7	0	8
Nail biting	0	0	0	0	0	0
Pallor	0	6	7	7	0	8
Eyes, dark circles	0	0	0	15	0	0
Total number of complaints	36	52	25	44	26	36
Total number of patients studied	15		13		12	

* Group A indicates children in whom pinworms were present on admission; group B, those who acquired pinworms after admission, and group C, those in whom pinworms were never present.

TABLE 3.—*Comparison of Symptoms Referable to Pinworm Infection in Children With and Without Infection, Obtained from Observers' Records Prior to Treatment*

Total number of patients	Positive for Pinworms 28		Negative for Pinworms 12	
Symptoms	Number	Per Cent	Number	Per Cent
Poor appetite	4	14	1	8
Abdominal pain	0	0	0	0
Underweight	9	29	3	25
Nausea and vomiting	0	0	0	0
Vaginitis	0	0	1	8
Restlessness	13	44	4	33
Irritability	8	28	6	50
Short attention span	15	33	7	53
Emotional instability	12	43	5	40
Hyperactivity	11	39	2	16
Insomnia	1	3	1	8
Enuresis	4	13	2	16
Encopresis	0	0	1	8
Masturbation	7	23	1	8
Listlessness	2	6	1	8
Nail biting	4	13	0	0
Pallor	2	6	0	0
Eyes, dark circles	2	6	1	8
No symptoms	3	10	1	8

tables 2 and 3 by comparing "parents' complaints" with "observers' remarks" prior to treatment. The observers reported a greater number of symptoms than did the parents in all groups. Parents and

observers both reported the highest incidence of symptoms in the group in which pinworms were present on admission. The incidence of symptoms reported by the observers for those who contracted pinworms in the hospital was comparable with that of the first group and significantly higher than in the children who never acquired the parasites.

For those who had pinworm on admission, outstanding symptoms reported by the parents were abdominal pain and insomnia, whereas the observers reported a high incidence of underweight, "nervousness," enuresis and masturbation. Poor appetite was common in both groups.

As for the group which acquired pinworms only after admission to the hospital, the symptoms were comparable throughout with those of the previous group, with a few exceptions. The parents did not report poor appetite or abdominal pain for these children. However, they listed a higher incidence of underweight and listlessness than they did for the others. The parents of the children who never had pinworms reported

TABLE 4.—*Effect of Treatment on Symptoms of Patients With and Without Oxyuriasis*

Type of Patients	Total No. of Patients	Per Cent Improved	Per Cent Worse	Per Cent Unimproved
With pinworms treated	14	21	7	72
With pinworms untreated	14	28	14	58
Without pinworms treated	7	14	25	61
Without pinworms untreated	5	20	20	60

primarily poor appetite and enuresis, whereas the observers stressed "nervousness" and underweight.

Effect of Treatment.—Table 4 shows the effect of treatment on the symptoms listed. In the group with pinworms who received treatment, 21 per cent improved symptomatically, as did 28 per cent of the children with pinworms who were untreated. In the same way, in the ones without pinworms, 14 per cent of those given treatment and 20 per cent of those untreated improved.

The corresponding figures for those becoming worse were 7, 25 and 20 per cent. The greatest number of all patients, regardless of classification, was finally listed as "unimproved."

In all the children treated with methyl rosaniline chloride, swabs for pinworm became negative and remained so for at least four weeks.

COMMENT

The general incidence of pinworm infection in this institution (70 per cent of a group of 40 children consecutively admitted) was found to be much higher than was suspected clinically. The incidence was

definitely greater than was recorded prior to the routine use of rigorous and repeated checks for the presence of the parasites. Many cases probably were missed in the past, and it is suspected that the occurrence of the condition generally in schools and institutions is higher than realized.

In comparison with other reports, symptoms referable to the gastrointestinal tract and perianal complaints were negligible in this study, whereas "nervous" symptoms were more numerous than in most. The latter observation may well be attributed to the type of patient resident here.

Suggestive Symptoms.—The symptoms of which parents complained were listed and differentiated in table 1 from those observed by the members of the hospital staff in the hope of identifying observations by parents which might be of value in the ordinary medical history. The nature of the patient population, however, may have affected the validity of these reports. The patients were all maladjusted children, with problems of long standing. In many cases the parents had become so emotionally involved as to develop unconsciously a generally negative and rejecting attitude. In such an atmosphere minor and even fancied evidences of ill health and abnormal behavior were easily seized on and exaggerated. This may explain discrepancies from the more objective observations and reports of members of the hospital staff.

The observations listed in tables 2 and 3 suggested the possibility that patients with pinworm had certain outstanding differences from those not harboring the parasite. The histories obtained from parents suggested a greater incidence of anorexia, underweight, enuresis, masturbation, pallor and listlessness in those patients who had pinworm prior to admission.

The reports from hospital observers indicated that among those who were infected with pinworm for a long period there was a greater incidence of anorexia and underweight. Enuresis was present in both old and recent infections, but it was possibly more pronounced in the former. Masturbation, pallor and listlessness were seen equally in both groups, while dark hollows under the eyes were noted only in those children with recent infection.

Some of the data given in table 2 were abstracted for further comparison in table 3. Here attention was directed solely to the observers' remarks for all children who had pinworms (acquired before or during the stay in the hospital) versus those who never were infected. The results emphasized the apparently more frequent occurrence in children with pinworm infection of the following symptoms: anorexia, hyperactivity, masturbation and nail biting.

Statistical analysis, however, with the use of the chi square test, showed that there was no statistically significant relationship between these symptoms and pinworm infection, even to the 10 per cent level of significance.

Effect of Treatment.—The effect of treatment was considered as a means of evaluating the significance of the various complaints. When studied symptom by symptom, none of these complaints showed any improvement attributable to the removal of the parasite. This study is summarized for the entire group in table 4.

Removal of pinworm infection in one group by means of anthelmintic therapy brought about no more improvement in suspected symptoms than occurred spontaneously in a similar group which received no therapy. This suggested that any apparently favorable changes could well be due to various nonspecific factors, such as the use of psychotherapy and environmental influences.

Since there was no clearcut evidence that any of the symptoms were specifically attributable to oxyuriasis and since there was no particularly demonstrable response of any symptom to specific therapy, it became the practice to withhold routine treatment for pinworm infection in this institution.

The medicinal treatment involved requires that the child regularly consume an appreciable number of pills for a succession of days, and these may have unpleasant side effects, such as nausea. If the parasite is to be eradicated, courses of treatment may need to be repeated several times. As the evidence for improvement is not impressive, it did not seem warranted to require the treatment routinely.

SUMMARY

1. Twenty-eight children infected with pinworm who were resident patients at a children's psychiatric hospital were studied and compared with 12 patients free of pinworm infection.

2. Symptoms noted with greatest frequency in pinworm-infected children were anorexia, hyperactivity, masturbation and nail biting. These did not appear significant on statistical analysis, however.

3. Perianal complaints were not observed and gastrointestinal symptoms were minimal.

4. Anthelmintic treatment had no specific effect in altering the symptom picture.

5. It is the current practice at this institution generally not to administer treatment to pinworm-infected patients.

Daniel V. Tramonti, B.S., performed the laboratory procedures in this study.

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Case Reports

RECURRENT PAROXYSMAL TACHYCARDIA IN A YOUNG INFANT

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AT THE Children's Free Hospital in Louisville and in the Children's Heart Clinic at the Louisville General Hospital, my associates and I have been particularly interested in the cardiac arrhythmias of infancy and childhood. In January 1946, Scott and I reported 2 cases of paroxysmal tachycardia in infants.[1] In November 1946, Prince, Ratondo and Scott [2] reported a case of auricular flutter and congenital heart disease, with restoration of normal rhythm by the use of quinidine. A series of cases of persistent extrasystoles in children was also studied.[3]

The case reported here of recurrent paroxysmal tachycardia in a young infant is unlike any of the cases of this disorder that I have seen described in the literature.

REPORT OF A CASE

Y. M. S., a 12 day old white female infant, was seen at home on Aug. 29, 1945, because of diarrhea, some vomiting, impetigo and fever. The infant was full term and was delivered by forceps on August 17, weighing 8 pounds 6½ ounces (3,813 Gm.). The mother was a primipara, but there were two half-sisters. One of these was said to have had a rapid heart rate in childhood. The paternal grandmother had a large toxic goiter and some cardiac involvement.

On examination, the infant's temperature was 101.4 F. rectally, and her heart rate was too rapid to be counted accurately. There was moderate dehydration and evidence of loss of weight. (On her admission to the hospital, her weight was 6 pounds 12 ounces [3,062 Gm.], or 1 pound 12 ounces [794 Gm.] under the weight at birth.) She also had severe impetigo, which had been treated with sulfathiazole ointment. There was no evidence of congestive failure. Paroxysmal tachycardia was suspected, and the infant was admitted to the Children's Free Hospital.

On her admission to the hospital, the heart rate was normal. Later the same evening, a house officer observed the infant in a paroxysm of tachycardia, but, by the time arrangements were made for the taking of an electrocardiogram, the rate was again normal. The following morning she again had an attack of tachycardia, which ceased spontaneously. The infant was hospitalized sixty-eight days. Paroxysms of tachycardia, with rates estimated by stethoscope of from 180 to 260, were recorded on forty-two different days, and some of the paroxysms were not

From the Children's Free Hospital and the University of Louisville School of Medicine.

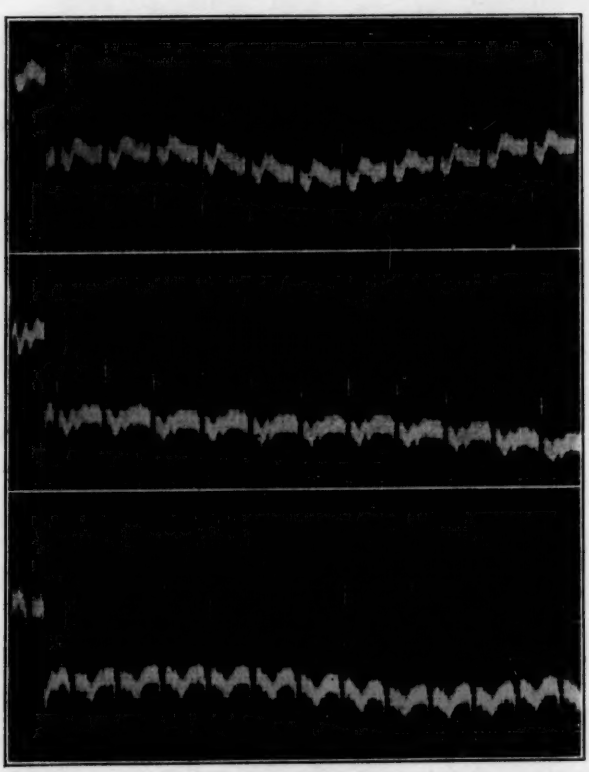
1. Scott, E. P., and Limper, M. A.: Paroxysmal Tachycardia: Report of Two Cases in Infants, J. Pediat. **28**:96 (Jan.) 1946.

2. Prince, G. E.; Ratondo, C. C., and Scott, E. P.: Auricular Flutter and Congenital Heart Disease, Am. J. Dis. Child. **72**:552 (Nov.) 1946.

3. Limper, M. A.: Extrasystoles in Children, to be published.

recorded. Several attacks lasted less than a minute, and the longest lasted about thirty-six hours. The infant did not show true congestive failure at any time, although there was slight edema of the eyelids occasionally, and she was somewhat dyspneic, pale and restless when she had prolonged seizures. Her general condition was poor during the first month in the hospital. Her diarrhea responded to treatment slowly, and she did not gain weight.

A blood count the day after her admission revealed a hemoglobin concentration of 17 Gm. per hundred cubic centimeters, 5,525,000 erythrocytes and 19,900 leukocytes, with 52 per cent polymorphonuclear forms and 48 per cent lymphocytes.



Electrocardiographic tracing made during a paroxysm when the heart rate was 220 in an infant with supraventricular paroxysmal tachycardia. Leads I to III are presented in order from top to bottom.

Three later white blood cell counts varied from 15,200 to 17,900, with 40 to 66 per cent polymorphonuclear forms. On one occasion, 8 per cent eosinophils were observed. The red blood cell count and hemoglobin concentration dropped gradually. On October 27, there were 3,600,000 erythrocytes, and the hemoglobin reading was 10 Gm. Results of three urinalyses were entirely normal. A Kahn test of the blood gave a negative reaction. A roentgenogram of the chest taken at a distance of 6 feet (183 cm.) revealed the heart to be normal in size and shape, and there was no evidence of pulmonary congestion. An electrocardiogram made on the night of the patient's admission was read as "probably normal for an infant of this age." The first tracing during a paroxysm of tachycardia was obtained

on September 1 and revealed supraventricular tachycardia, with a rate of 240 per minute. A diphasic T wave in lead I and inverted T waves in Leads II and III were present. The figure is a reproduction of a tracing taken during a paroxysm on September 6, when the rate was 220. A tracing on October 16 revealed a rate of 290 but was unsuitable for reproduction.

The patient's diarrhea was treated with diet and parenteral administration of fluids. After the diarrhea was under control, and anemia had developed, she was given a liquid ferrous sulfate preparation orally and injections of a liver extract concentrate parenterally. The impetigo was treated with gentian violet and ultraviolet radiation.

An attempt was made to control the tachycardia with intramuscular and oral administration of digitalis U. S. P., at first in intermittent doses and later by digitalization and maintenance therapy. Single doses consisted of ½ cat unit intramuscularly. In one instance, an overdose of digitalis was ordered through an error, the order reading, "⅓ cat unit every three hours," instead of "⅓ cat unit three times a day." Clinically, no toxic effect was noted, and the paroxysms continued.

On the eighth day in the hospital, September 6, administration of quinidine sulfate U. S. P., in a dosage of ½ grain (32 mg.) every six hours, was started. The dosage was later increased to ¾ grain (48 mg.) every six hours, and during one twenty-four-hour period 1 grain (65 mg.) was given every six hours. The drug was discontinued on September 22 and resumed on October 20; from that date to October 21, the dosage was ½ to ¾ grain every four hours six times a day.

On three occasions, 0.1 mg. of cedilanid® (lanatoside C, U. S. P., a glycoside obtained from digitalis) was given intravenously, with dramatic reduction in the heart rate, associated, however, with some pallor and transient cyanosis. No practical method of giving a drug of this type prophylactically, as was suggested by Tandowsky[4] for adults, was worked out for this patient. Fortunately, the infant improved spontaneously. Her paroxysms became less frequent and of shorter duration, and she began gaining weight. It was possible to diagnose paroxysms of tachycardia by the pulsation of the neck vessels. The mother was instructed to look for this sign when the infant was discharged from the hospital on November 6. She was quite sure that several seizures occurred at home; vomiting and restlessness usually accompanied them. The infant was given phenobarbital elixir U. S. P., and the attacks finally ceased.

The patient has been under regular observation since that time and has shown no signs of a congenital anomaly of the heart. Her heart rate under resting conditions is normal. One attempt was made to obtain an electrocardiogram, but the infant screamed and was so uncooperative that the attempt was abandoned. Her muscular and mental development has been somewhat precocious.

COMMENT

The usual picture of paroxysmal tachycardia in infancy is that of digestive disturbance and fever, followed by grayish pallor, dyspnea and signs of congestive failure. Recurrent attacks of paroxysmal tachycardia in infants were reported by Hubbard[5] and others. Four of

4. Tandowsky, R. M.: Prophylactic Use of Lanatoside C in Auricular Paroxysmal Arrhythmias, Am. Heart J. **29**:71 (Jan.) 1945.

5. Hubbard, J. P.: Paroxysmal Tachycardia and Its Treatment in Young Infants, Am. J. Dis. Child. **61**:687 (April) 1941.

Hubbard's 9 patients had from one to several attacks, diagnosed clinically or on routine electrocardiographic study within a few days or up to six weeks after the initial episode. Farr and Wegman [6] reported a case of extreme tachycardia in an infant; they were uncertain whether the condition represented paroxysmal tachycardia, or whether it might be one of auricular flutter with a 1:1 ratio. The first attack occurred at the age of 23 days and the sixth at 8½ months. They were of the opinion that digitalis therapy did not have much influence on the course, but, in reading the report, one is impressed with the fact that the attacks subsided soon after digitalis was given. Hobbs [7] reported on an infant, delivered by mid forceps after a long labor, with seven attacks of paroxysmal tachycardia, the first of which occurred at 2 months and the last at about 6½ months of age.

Almost as unusual as the number and frequency of attacks in the patient here reported on was the failure to respond to adequate doses of digitalis given orally and intramuscularly or to quinidine sulfate. Among the cases that Hubbard [5] collected from the literature, only 1 was of a patient treated with an adequate dose of digitalis who failed to survive. This was the patient of Wegman and Egbert,[8] an infant who was observed at autopsy to have a rhabdomyoma of the heart. Hubbard's own patients responded satisfactorily to digitalis. Since the publication of Hubbard's report, Young [9] reported a case of paroxysmal tachycardia (rate, 310 electrocardiographically) in a 25 day old infant, who died of congestive heart failure in spite of medication with digitalis and moryl® (carbaminoylcholine chloride). Autopsy revealed cardiac enlargement and a patent ductus arteriosus, but it would seem that the latter is not especially significant in an infant of this age. In addition to congestion of the viscera, an excess of serous fluid was observed in the pericardium and pleural cavities.

Hubbard and Starbuck [10] in 1943 reported on an infant with extreme supraventricular tachycardia who was treated with digifolin® intravenously and intramuscularly; the paroxysm subsided after the intramuscular injection, as did a second paroxysm twelve hours later. The infant here reported on is the second with paroxysmal tachycardia who was treated with intravenously administered digitalis. Treatment of this patient was only temporarily successful.

Gold [11] recommended using quinidine before trying digitalis in paroxysmal auricular tachycardia, because digitalis is excreted slowly,

6. Farr, L. E., and Wegman, M. E.: Extreme Tachycardia in the Newborn, Am. J. M. Sc. **190**:22 (July) 1935.

7. Hobbs, L. F.: Paroxysmal Tachycardia in Infancy, Am. Heart J. **21**:804 (June) 1941.

8. Wegman, M. E., and Egbert, D. S.: Congenital Rhabdomyoma of the Heart Associated with Arrhythmia, J. Pediat. **6**:818 (June) 1935.

9. Young, J. H.: Paroxysmal Tachycardia in an Infant, M. J. Australia **1**:538 (June 10) 1944.

10. Hubbard, J. P., and Starbuck, G. W.: Paroxysmal Tachycardia in a Two Month Old Infant with a Ventricular Rate of 350, Am. J. Dis. Child. **65**:582 (April) 1943.

11. Gold, H.: Treatment of Cardiac Arrhythmias, M. Clin. North America **24**:577 (May) 1940.

and the two drugs should not be used simultaneously. The use of combined therapy is apparently a common practice in the treatment of adults, however.

As in cases previously described, the causation of the tachycardia in the patient here reported on is unknown. Both this patient and that seen by me in 1944 [1] had had impetigo and had been treated with a sulfonamide preparation locally, but this is probably merely a coincidence. Several authors expressed the opinion that infection is a factor in the causation of paroxysmal tachycardia in infants. The mother of Young's patient had a history of some type of exanthem when three months pregnant. In the patient here reported on it was difficult to determine whether the diarrhea was a result, or a possible cause, of the paroxysmal tachycardia. The patients of Hobbs and Hubbard and Starbuck had a history of difficult delivery, and in Hobbs's patient this followed a long labor. My patient had no such history.

Dr. W. C. Gettelfinger, the cardiologist who saw this patient and interpreted the electrocardiograms, and I feel that the disorder observed in this infant was much more like the paroxysmal tachycardia seen in older children and adults than like that usually observed in early infancy. Frequently recurring attacks of short duration which do not always respond to digitalis or quinidine seem to be fairly common in persons of the older age group. None of the patients with paroxysmal tachycardia in infancy studied in the literature presented the picture seen in this patient.

SUMMARY

An unusual case of paroxysmal tachycardia in a young infant is reported. It is postulated that the infant exhibited the "adult type" of this disorder, rather than the usual picture observed in infancy. The literature pertinent to this report is briefly reviewed.

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OSTEOMA CUTIS

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AND
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CASES of osteoma cutis have been reported under various titles, such as osteosis cutis, osteomatosis cutis, miliary osteoma and disseminated congenital osteomas. Many of the lesions are secondary ossifications, according to Borst,[1] and may have originated from injuries, inflammatory processes or tumors. Recently, Vero, Machacek and Bartlett [2] gave a good, complete review of the literature.

A case of this disease seems to be of sufficient rarity to record in the medical literature.

REPORT OF A CASE

K. J., a 9 year old white boy, was admitted to the Children's Free Hospital, May 1, 1947, with the chief complaints of draining ears, obesity and cutaneous trouble. The present illness dated from early infancy.

At birth, he weighed 10 pounds (4,536 Gm.) and was apparently normal. He gained weight rapidly, and on Aug. 25, 1938, at the age of 5 months, he was admitted to the Vanderbilt University Hospital, Nashville, Tenn., with a weight of 32 pounds 10½ ounces (14.8 Kg.). At this time, the circumference of his head was 18 inches (45.7 cm.), of his chest 22 inches (55.9 cm.) and of his abdomen 22 inches; his height was 29 inches (73.7 cm.). According to the parents, he had a vesicular eruption on the right thigh at 2 months of age. The rash spread rapidly and formed hard, firm, nontender lesions in the skin. Subsequently, there appeared similar lesions on the arms, fingers, abdomen and left leg.

The physical examination at this time revealed a large obese child with hard, roughened areas on the skin as described, the largest, in the thigh, being 2 by 4 cm. The fontanel measured 2 by 2 cm., with no evidence of increased pressure. Except for bilateral otitis media, results of the remainder of the physical examination were not remarkable.

Laboratory studies at this time revealed 22,000 white blood cells per cubic millimeter and negative reactions to the tuberculin and Kahn tests. Roentgenograms of the skull showed no abnormality. The dextrose tolerance test revealed a normal curve; serum calcium measured 10.1 mg. per hundred cubic centimeters. The

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From the Department of Pediatrics, University of Louisville School of Medicine, service of James W. Bruce, M.D., and W. W. Nicholson, M.D.

1. Borst, M., in Aschoff, K. A. L.: Pathologische Anatomie, Jena, Gustav Fischer, 1928, vol. 1, p. 728.

2. Vero, F.; Machacek, G. F., and Bartlett, F. H.: Disseminated Congenital Osteomas of Skin, with Subsequent Development of Myositis Ossificans: Report of Case in Infant, J. A. M. A. **129**:728 (Nov. 10) 1945.

parents were uncooperative, and, despite a temperature of 103 F., the child was permitted to leave the hospital August 28, with a diagnosis of pituitary obesity, scleroderma and suppurative acute bilateral otitis media.

He was next seen June 17, 1939 at the Vanderbilt Clinic "for an examination for a fair of some sort." At this time he weighed 67 pounds (30.4 Kg.) and was only 14 months of age.

Fig. 1.—Photograph of child with osteoma cutis at the time of his admission to the hospital.

Fig. 2.—Photograph showing chief site of distribution of osteomas, superior to the left internal malleolus.

On Nov. 26, 1943 he again returned to the Vanderbilt Clinic because of generalized convulsions which began three years previously, the last being seven weeks prior to his admission to the outpatient department. Frontal headaches had developed since his previous admission. A complete physical examination revealed no signs of increased intracranial pressure. His blood pressure was 110 systolic and 70 diastolic, and his weight had increased only to 68 pounds (30.8 Kg.).

Fig. 3.—Roentgenogram (top) showing small areas of apparent calcification in the right thigh and left ankle. The middle roentgenogram is a view from above the ankle region and the bottom film a view from the side.

From his past history it was learned that he sat alone at 6 months. Dentition began at 7 months, but he did not walk until 3 years of age. Although 9 years of age, as yet he had not started to go to school and could neither read nor write. It was reported that he was enuretic and a sleep walker. With the exception of one stillborn sibling, the family history and the remaining past history were not contributory.

The physical examination on his admission to the Children's Free Hospital revealed a temperature of 100 F. The appearance was that of an obese white

Fig. 4.—Photomicrograph of tissue taken for biopsy from the lesion on the thigh varifying the diagnosis of osteoma cutis.

boy (fig. 1) who was much withdrawn and difficult to examine. The positive physical findings consisted of purulent otitis media on the left, nonsuppurative otitis media on the right, a sty of the right eye and hard individual masses located beneath the skin and distributed over the entire body but chiefly in the area of the right groin and just superior to the left internal malleolus (fig. 2). These lesions were nontender and quite adherent to the overlying skin but not to the underlying tissues. They were freely movable and of various sizes up to 12 cm. in length.

Laboratory examination revealed a white blood cell count of 22,050, with 59 per cent polymorphonuclear leukocytes, 32 per cent lymphocytes, 2 per cent basophils and 4 per cent eosinophils. Examination of the urine showed a 2 plus reaction for albumin, with 5 to 15 red blood cells per high power field. Subsequent urinalyses gave entirely normal results. The roentgenograms revealed small areas of apparent calcification in the regions of the right thigh and left ankle (fig. 3) and hilar enlargement (4 plus) with heavy markings in each lung. A pneumoencephalogram displayed no abnormalities. Determinations revealed 7.1 mg. of calcium and 13.1 mg. of phosphorus per hundred cubic centimeters of blood. The blood cholesterol measured 224.7 mg. per hundred cubic centimeters. Alkaline phosphatase was 8.0 Bodansky units. Reactions to the Kahn test of the blood and to the intradermal tests with old tuberculin (0.1 mg.) and histoplasmin (2.0 mg.) were all negative.

On the sixth day in the hospital a biopsy of the lesion on the thigh previously described was done. The microscopic diagnosis was osteoma cutis (fig. 4).

The otitis media responded to local irrigations of warm sterile isotonic sodium chloride solution U. S. P. and penicillin, 50,000 units given intramuscularly every three hours for six days.

At the request of the parents, he was discharged on the eleventh day from the hospital, with the diagnosis of osteoma cutis and mental retardation.

He was readmitted to the Children's Free Hospital, Aug. 26, 1947 because of convulsions. The seizures began one week prior to the admission with one convulsion. The following day there was another. Five days before admission there were five generalized seizures. The day before his admission four convulsions occurred. The convulsions were spastic in nature, involving first the upper extremities, then the whole body, each seizure lasting three to four minutes. No fever, vomiting or other neurologic disturbances had been noted.

The physical examination at this time revealed nonsuppurative otitis media on the right. The subcutaneous masses previously described were still present. His weight had increased to 91 pounds (41.3 Kg.).

Complete laboratory studies were again performed and revealed the cerebrospinal fluid to be normal. Blood calcium measured 9.1 mg., blood phosphorus, 9.4 mg., and blood cholesterol, 236.7 mg., per hundred cubic centimeters. Alkaline phosphatase was 15.4 Bodansky units. The red and white blood cell counts and the results of urinalysis were normal. Roentgenograms of the mastoid area revealed sclerosis of the antral and periantral regions bilaterally.

The course in the hospital was uneventful and treatment was only symptomatic. After ten days of observation without convulsive seizures, he was again discharged.

SUMMARY

A case of osteoma cutis in a 9 year old child is reported.

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Progress in Pediatrics

PLASMA VITAMIN A AND ITS CLINICAL SIGNIFICANCE

A Review

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THE DEVELOPMENT of reliable methods for the determination of vitamin A in the blood plasma or serum has greatly contributed to the knowledge of the function of vitamin A in the human organism. During the past five years my associates and I have published certain studies on plasma vitamin A.[1] I wish to present here some of these results, together with other reports from the literature which, I believe, are of interest to the pediatrician.

VITAMIN A IN THE HUMAN BODY

The more our studies progressed the more it became evident that the plasma vitamin A is largely independent of the dietary intake of vitamin A or its precursor carotene. In several nutritional surveys, it was reported that the values for plasma vitamin A for well nourished persons are on a higher level than those for poorly nourished ones.[2] We observed that the plasma vitamin A values of persons from the more favorable socioeconomic groups are, on an average, higher than the values for persons from the less favorable groups. Under the nutritional conditions prevailing in this country the differences between well and poorly nourished persons are less pronounced than the differences among various groups of persons under physiologic conditions.

The plasma vitamin A of the newborn infant is approximately half that of a normal adult and is completely independent of the plasma vitamin A of its mother or the mother's oral intake of vitamin A

1. (*a*) Abt, A. F.; Aron, H. C. S.; Bimmerle, J. F.; Bundesen, H. N.; Greenebaum, R. S.; Fagen, H. J.; Farmer, C. F.; Wenger, O. C., and White, J. L.: Quart. Bull., Northwestern Univ. M. School **16**:241-245, 1942. (*b*) Aron, H. C. S.: Nebraska M. J. **30**:1-5, 1945. (*c*) Aron, H. C. S.; Craig, R. M.; Farmer, C. F.; Kendell, H. W., and Schwemlein, G. X.: Proc. Soc. Exper. Biol. & Med. **61**:271-276, 1946.

2. Darby, W. J., and Milam, D. F.: Am. J. Pub. Health **35**:1014-1021, 1945. Harris, P. L.; Hickman, K. C. D.; Jensen, J. L., and Spies, T. D.: ibid. **36**:155-160, 1946. Shank, R. E.; Coburn, A. F.; Moore, L. V., and Hoagland, C. L.: J. Clin. Investigation **23**:289-295, 1944.

during pregnancy (Lund and Kimble).[3] The plasma vitamin A remains at this low level until about the fourth day of life when it shows a definite and sudden rise (Lewis, Bodansky and Shapiro[4]). We found that children under 16 years of age, on the average, have lower values for plasma vitamin A than the adults of the same families. Male adults have definitely higher plasma vitamin A values than female adults.[5] Pregnant women have lower plasma vitamin A values than nonpregnant women.[6] The depression of the plasma vitamin A is most pronounced during the last three months of pregnancy. Without any change in the diet, even after withdrawal of vitamin A supplements which were given during pregnancy, we observed a definite rise of the plasma vitamin A after delivery with nearly every woman examined.[1a] Lund and Kimble showed that the rise of plasma vitamin A is complete twenty-four to forty-eight hours after delivery.[3]

Under normal conditions, the human organism has the tendency to maintain its plasma vitamin A at a constant level which is characteristic for the individual person. This is impressively revealed by studies of the plasma vitamin A after increased and after restricted oral intake of vitamin A. After a single large oral dose of vitamin A, a temporary rise of the plasma vitamin A is recorded in three to six hours, returning in less than twenty-four hours to the original level (fig. 1). Daily supplements of 50,000 international units of vitamin A given to healthy nurses for as long as twenty-two months did not raise the plasma vitamin A except for a few hours after intake (Robertson and Morgan[7]). Lewis, Bodansky and Haig[8] reported that daily supplements of 17,000 international units of vitamin A given to young children, 6 to 18 months old, had no lasting effect on the vitamin A content of the blood. We gave 250,000 international units of vitamin A daily to a healthy volunteer. After twelve days, with a total intake of 3,000,000 international units of vitamin A, the plasma vitamin A became nearly twice as high as it had been before supplementation. But when the supple-

3. Lund, C. J., and Kimble, M. S.: Am. J. Obst. & Gynec. **46**:207-221 and 486-501, 1943.

4. Lewis, J. M.; Bodansky, O., and Shapiro, L. M.: Regulation of Level of Vitamin A in Blood of Newborn Infants, Am. J. Dis. Child. **66**:503-510 (Nov.) 1943.

5. Kimble, M. S.: J. Lab. & Clin. Med. **24**:1055-1065, 1939. Abt and others.[1a]

6. Bodansky, O.; Lewis, J. M., and Lillienfeld, M. C. C.: J. Clin. Investigation **22**:643-647, 1943. Abt and others.[1a] Lund and Kimble.[3]

7. Robertson, E. C., and Morgan, A. L.: J. Nutrition **31**:471-484, 1946.

8. Lewis, J. M.; Bodansky, O., and Haig, C.: Level of Vitamin A in Blood as Index of Vitamin A Deficiency in Infants and in Children, Am. J. Dis. Child. **62**:1129-1148 (Dec.) 1941.

ments were discontinued the original plasma level was reestablished within ten days (fig. 2).

On the other hand, when the oral intake of vitamin A and carotene in the diet is greatly reduced or practically nil, the plasma vitamin A may remain unchanged for several months.[9] The human organism

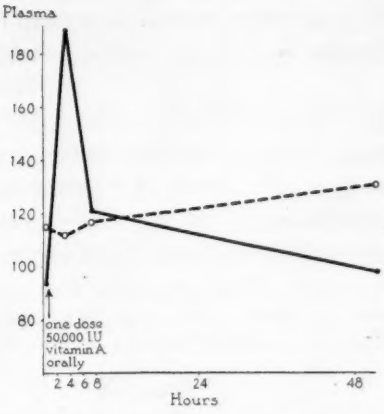

Fig. 1.—Plasma vitamin A and carotene levels after a single oral dose of vitamin A. The solid line indicates the number of international units of vitamin A, while the broken line indicates the micrograms of carotene, per hundred cubic centimeters of plasma (from Aron[1b]).

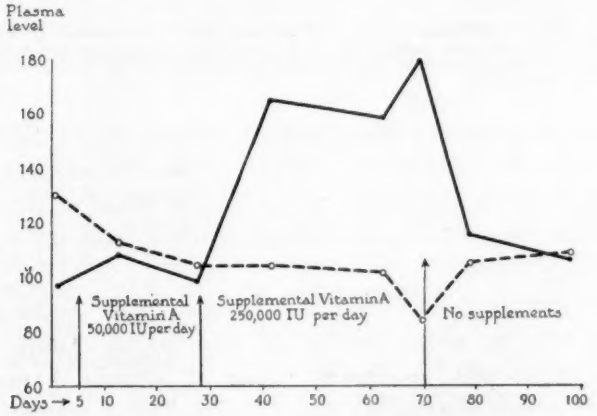

Fig. 2.—Plasma vitamin A and carotene levels after various orally administered supplements of vitamin A. The solid line indicates the number of international units of vitamin A and the broken line, the micrograms of carotene, per hundred cubic centimeters of plasma (from Aron[1b]).

9. Brenner, S., and Roberts, L. I.: Effects of Vitamin A Depletion in Young Adults, Arch. Int. Med. **71**:474-482 (April) 1943. Wagner, K. H.: Ztschr. f. klin. Med. **137**:639-647, 1940. Wald, G.; Brouha, L., and Johnson, R. E.: Am. J. Physiol. **137**:551-556, 1942.

has at its disposal large reserves of vitamin A, comparatively much more than of any other vitamin. Approximately 85 to 95 per cent of these reserves are stored in the liver, the remainder in bone marrow, kidney and other tissue.[10] The liver of a well nourished adult contains several hundred times as much vitamin A as is in the circulating blood. The vitamin A reserves of young children may be comparatively smaller, but even in the liver of the newborn there is present from 50 to 100 times as much vitamin A as in the circulating blood (table).

MAINTENANCE OF VITAMIN A LEVELS

The plasma vitamin A level is maintained by means of the reserves of vitamin A in the liver. An excess of vitamin A is deposited here.[11] When the intake of vitamin A and carotene is insufficient, vitamin A from the liver is released into the blood. In this way the vitamin A level

Partition of Vitamin A Between Blood and Liver *

Age	Total in Blood Plasma, I. U.	Total in Liver, I. U.
Newborn	100 to 150	15,000 (4,000 to 40,000)
1 yr	450 to 600	80,000 (50,000 to 120,000)
10 yr	1,100 to 1,300	250,000 (120,000 to 500,000)
Adult	3,200 to 4,000	1,000,000 (400,000 to 4,000,000)

* Abt and others.[1a] Williams.[10] Ellison, J. B., and Moore, T.: Biochem. J. **31**: 165-171, 1937. Henley, T. H.; Dann, M., and Golden, W. R. S.: Reserves, Absorption and Plasma Levels of Vitamin A in Premature Infants, Am. J. Dis. Child. **68**: 257-264 (Oct.) 1944. Moore, T.: Biochem. J. **31**: 155-164, 1937.

in the blood is maintained fairly constant, independent of the oral intake. Disturbance in the conversion of the provitamin carotene into vitamin A is equivalent to a diminished or an insufficient intake of vitamin A and must be compensated for by release of vitamin A from the reserves into the circulating blood.

A significant decline of the plasma vitamin A can be caused by two conditions: (1) an exhaustion of the reserves of vitamin A (in the liver) combined with insufficient intake of vitamin A and carotene (prolonged severe starvation) and (2) insufficient release of vitamin

10. Williams, R. J., in Harris, R. S., and Thimann, K. K.: Vitamins and Hormones: Advances in Research and Applications, New York, Academic Press, Inc., 1943, vol. 1, pp. 229-247.

11 (*a*) Abels, J. C.; Gotham, A. T.; Pack, G. T., and Rhoads, C. P.: J. Clin. Investigation **20**:749-764, 1941. (*b*) Ralli, E. P.; Papper, E.; Paley, K., and Bauman, E.: Vitamin E_α and Carotene Content of Human Liver in Normal and in Diseased Subjects: Analysis of One Hundred and Sixteen Human Livers, Arch. Int. Med. **68**:102-111 (July) 1941.

A from the reserves (in the liver) into the circulating blood (by a disturbance of the regulatory mechanism). Complete exhaustion of the reserves in the liver requires many months, if not years, of severe malnutrition. After the first World War we were able to demonstrate the absence of vitamin A in the extracts prepared from livers of infants who succumbed from undernutrition with definite keratomalacia. A recently published report [12] revealed that in some Dutch children after prolonged severe starvation very low, even 0, values for plasma vitamin A were demonstrated.

In this country exhaustion of the vitamin A reserves of the body will hardly ever be observed. When definite changes in the vitamin A content of the blood are seen here, they are practically always caused by some disturbance of the metabolic regulation of the vitamin, by an insufficient release of the vitamin from the liver into the blood. It is the recognition of this fact which makes the study of the plasma vitamin A a problem of clinical interest.

VITAMIN A IN HEPATIC DISEASE

Since the liver is the principal storage organ of the body, it is not surprising that in various types of hepatic diseases abnormally low values for plasma vitamin A are found.[13] In acute hepatitis, the depression of the plasma vitamin A is observed several days before accumulation of bilirubin in the serum, and the icterus index becomes pathologically high.[14] From the slope of the curve for plasma vitamin A, we were able in several cases to predict the development of jaundice which followed two or three days later.[1c] Conversely, the plasma vitamin A rises as soon as the hepatic function returns to normal, much earlier than the icterus index goes down. Bilirubin remains in the serum, leaving a "residual pigmentation" for quite some time, in spite of the fact that the liver is functioning normally again. The change in the level of the plasma vitamin A is a valuable and sensitive indicator of hepatic function. How far the determination of plasma vitamin A will develop into a generally applicable test for hepatic function is a technical and methodologic problem. Clinically, these observations explain how, in the course of long-standing impairment of the hepatic

12. Jonxis, J. H. P.: Nutrition Rev. **4**:97-99, 1946.

13. Footnote 11. Meyer, K. A.; Steigmann, F.; Popper, H., and Walters, H. W.: Influence of Hepatic Function on Metabolism of Vitamin A, Arch. Surg. **47**:26-43 (July) 1943. Ralli, E. P.; Bauman, E., and Roberts, L. B.: J. Clin. Investigation **20**:709-713, 1941.

14. Popper, H.; Steigmann, F.; Meyer, K. A., and Zevin, S. S.: Relation Between Hepatic and Plasma Concentrations of Vitamin A in Human Beings, Arch. Int. Med. **72**:439-460 (Oct.) 1943. Popper, H.; Steigmann, F., and Zevin, S. S.: J. Clin. Investigation **22**:775-783, 1943.

function, symptoms of vitamin A deficiency may develop. When continuously insufficient vitamin A is released into the blood, the tissues will obtain less vitamin A than is required for their normal activity; symptoms of lack of vitamin A, e. g., night blindness, may eventually develop.

VITAMIN A LEVELS IN FEBRILE STATES

For many years the interest of pediatricians was attracted by the fact that extremely low values of plasma vitamin A were recorded during febrile infectious diseases, especially during pneumonia. The Swedish pediatrician, Lindqvist,[15] analyzed the livers of children who had died of pneumonia. While the plasma vitamin A during the last days before death had been very low, in some of the children even 0, the stores in the liver were discovered full of vitamin A. These observations furnished definite proof that the depression of the plasma vitamin A during the febrile state of pneumonia is caused by a blockade of vitamin A in the liver.

One question still remained open: What causes the depression of the plasma vitamin A during febrile infectious diseases? Is it the infectious process as such, or is it the elevation of the body temperature to febrile height? This problem could best be solved by following the plasma vitamin A when the body temperature was elevated to febrile height, not by an infectious process but by physical means.

The results of such studies were published from the Chicago Intensive Treatment Center.[1c] Here, fever physically induced by the Kettering hypertherm® in combination with chemotherapy is used as a method of intensive treatment of syphilis and gonorrhea. It appeared pertinent for the safe conduct of this combined therapy to find a biochemical indicator which might serve as a measurement of the intensity of the action of physically induced fever on the human organism. Since no other biochemical determination had been found which might serve this purpose, it was suggested that a systematic study of the plasma vitamin A under the influence of induced fever might furnish valuable information. The results obtained surpassed our expectations.

The studies,[1c] including 111 patients, showed that elevation of the body temperature to 105.5 or 106 F. (rectal) was followed by a depression of the plasma vitamin A which lasted until the first morning after fever. On the second morning after fever a decided rise of the plasma vitamin A was recorded, and on the third morning the plasma vitamin A had returned approximately to the prefever level. This restoration took place spontaneously without any special medication or dietary supplements. The changes in the plasma vitamin A were most pro-

15. Lindqvist, T.: Klin. Wchnschr. **16**:1345-1348, 1937; Acta med. Scandinav., 1938, supp. 97, pp. 1-262.

nounced in those patients receiving physically induced fever and chemotherapy of eight hours' duration. The course of the plasma vitamin A recorded for each individual patient in international units per hundred cubic centimeters of plasma is demonstrated in figure 3. There

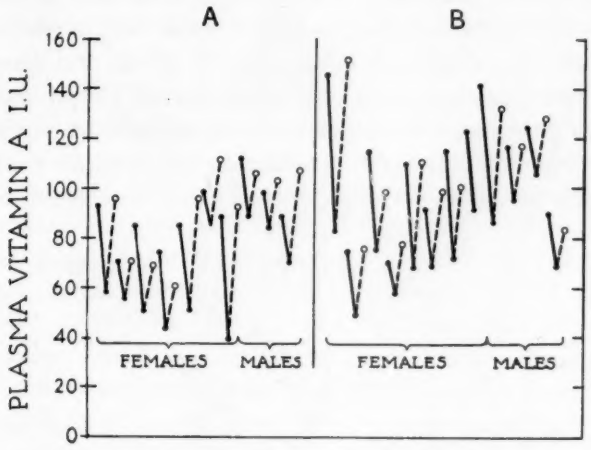

Fig. 3.—Decline of plasma vitamin A levels during physically induced fever and their rise after fever and chemotherapy of eight hours' duration. Data in portion A are for patients whose temperatures were raised to 105.5 F. Before fever they received bismuth and during fever, oxophenarsine hydrochloride (mapharsen®). Portion B shows data for patients whose temperatures were raised to 106 F. They received sulfonamide compounds before fever and no drug during fever. The unbroken line indicates a decline in plasma vitamin A levels and the broken line a rise in the levels (from Aron and others[1c]).

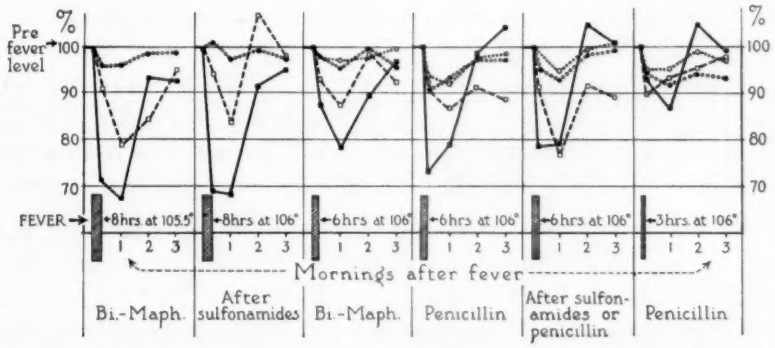

Fig. 4.—Medians for plasma vitamin A, carotene, hemoglobin and protein in percentage of prefever levels. The unbroken line indicates levels for vitamin A, the broken line, for carotene, the lines of open circles, for hemoglobin, and the lines of solid circles for plasma protein (from Aron and others [1c]).

are some variations in the extent of these changes, but the pattern is always the same, i. e., a notable decline after the physically induced fever and chemotherapy, followed by a rapid spontaneous return toward, or even over, the prefever level.

The next figure (fig. 4) demonstrates that the course of the plasma vitamin A was almost identical, irrespective of the chemotherapeutic agents given before or during physically induced fever (bismuth subsalicylate, oxophenarsine hydrochloride [mapharsen], sulfathiazole and the sodium salt of penicillin). In this figure, the values are given in per cent of the prefever level; the graphs show the "medians" for each group of patients. The next figure (fig. 5) shows the "medians" for vitamin A, carotene and total plasma protein of the patients grouped according to the number of hours their body temperature was maintained at 105.5 to 106 F. This graph reveals that the extent of the depression of the plasma vitamin A is directly related to the duration of fever. The

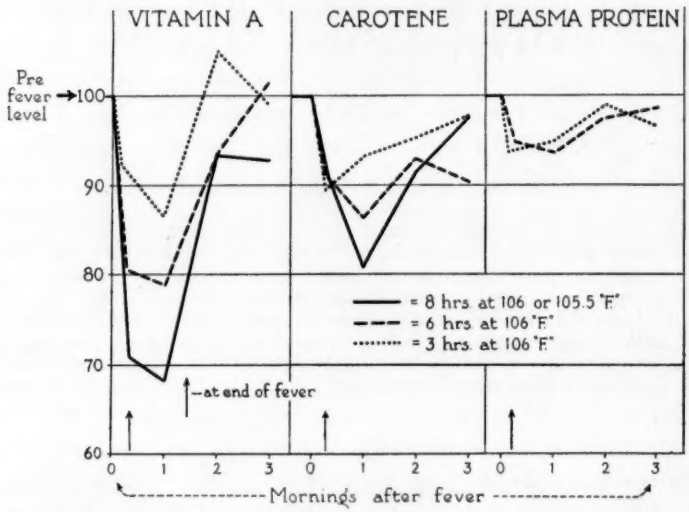

Fig. 5.—Graph showing medians for vitamin A, carotene and plasma protein, in percentage of prefever levels, of patients treated with physically induced fever and chemotherapy. Levels are shown after eight, six and three hours of fever at 105.5 or 106 F. (from Aron and others [1c]).

plasma vitamin A level drops approximately 30 per cent after eight hours, 20 per cent after six hours and 10 per cent after three hours of fever at 106 F.

These findings can be regarded as a proof that elevation of the body temperature to 105.5 and higher, not by an infectious process but by physical means, is accompanied by a definite impairment of the metabolic regulation of vitamin A as revealed by the insufficient release of the vitamin, from the hepatic stores into the circulating blood. Elevation of the body temperature for six hours definitely affects the plasma vitamin A level more than severe restriction of the dietary vitamin A intake for six months.

RESULTS OF VITAMIN A TOLERANCE TESTS

Several interesting facts have been elicited by vitamin A tolerance tests. In such tests, doses of from 1,000 to 7,500 international units of vitamin A per kilogram of body weight are given orally and the course of the plasma vitamin A is followed. Under normal conditions, the peak of the rise of the plasma vitamin A is seen after three to six hours, as was shown previously in figure 1. An insufficient rise in the plasma vitamin A, the "flat vitamin A curve," is a well known diagnostic sign of fibrocystic and of celiac disease[16]; similar "flat vitamin A curves" were reported in patients with sprue[17] or with long-standing colitis.[18] It is generally assumed that these flat vitamin A curves are caused by poor intestinal absorption of vitamin A.[19] But flat vitamin A curves have also been recorded in hepatic diseases, especially in hepatic cirrhosis and, as will be discussed afterward, in certain disorders of the skin. These observations make it disputable whether the flat curves for plasma vitamin A are always an expression of poor intestinal absorption. It should be kept in mind that insufficient release of vitamin A into the blood, impairment of the regulatory mechanism, can produce similar flat vitamin A curves. The beneficial effect of crude liver extract on the curves for vitamin A in celiac disease makes one think in the same direction.

VITAMIN A DEFICIENCY DISEASES

Last, the question of the plasma vitamin A in so-called vitamin A deficiency diseases will be briefly discussed. It has already been emphasized that the severe types of vitamin A deficiencies, as keratomalacia or xerosis corneae, are rarely observed any more in the Chicago area. What are seen here nowadays are sometimes obstinate, though mild, symptoms of vitamin A deficiency, as follicular conjunctivitis and disorders of the skin, ranking from dryness and roughness to pityriasis rubra pilaris and keratosis follicularis. In some instances, it is possible to prove that the patients who exhibit these symptoms, for some reason or other, have taken a diet exceedingly low in vitamin A for a long time. Such patients usually show definite improvement of their clinical symptoms when an adequate diet and ample vitamin A supplements are given. However, from the reports in the literature and from personal experience, it appears that in many, if not in the majority, of cases of

16. May, C. D.; McCreary, J. F., and Blackfan, K. D.: J. Pediat. **21**:289-305, 1942.

17. Cayer, D.; Ruffin, J. M., and Perlzweig, W. A.: Am. J. M. Sc. **210**:200-207, 1945.

18. Page, R. C., and Bercovitz, Z.: Am. J. Digest. Dis. **10**:174-177, 1943.

19. Adlersberg, D.; Sobotka, H., and Bogatin, B.: Gastroenterology **4**:164-174, 1945.

follicular conjunctivitis,[20] pityriasis rubra pilaris [21] and keratosis follicularis, no history of insufficient vitamin A intake can be ascertained.[22]

Several authors [23] reported that daily supplements of from 25,000 to 100,000 international units of vitamin A given over a period of several months had no curative effect with most of these patients. Only when doses of vitamin A of 200,000 international units and more were given was temporary improvement of the cutaneous disorders reported in some patients (Peck, Glick, Sobotka and Chargin).[24] However, when these excessive vitamin A supplements were reduced or discontinued, the cutaneous symptoms reappeared. The excretion of vitamin A in the feces was normal; thus, insufficient intestinal absorption could not be the underlying cause.

Determinations of the plasma vitamin A level of the aforementioned patients gave values within the normal range. We made similar observations on children who had definite follicular conjunctivitis. Remarkable results were reported when the patients with cutaneous disorders, mentioned previously, were given large doses of vitamin A. After one massive oral dose of vitamin A, abnormally flat curves for vitamin A were recorded by Ruch, Brunsting and Osterberg.[25] When large doses of vitamin A were administered for several weeks, the elevation of the plasma vitamin A levels of the patients with skin disorders was definitely smaller than in normal persons after comparable oral doses of vitamin A (Peck and his associates [24]).

From this behavior of the plasma vitamin A level, it can be concluded that the symptoms of vitamin A deficiency are caused by a disturbance of the regulatory mechanism for the vitamin, the inability to release sufficient vitamin A into the blood. It appears justifiable to follow Sulzberger's suggestion and to designate these cutaneous disorders as dysvitaminoses, not as avitaminoses.

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20. Sandels, M. R.; Cate, H. D.; Wilkinson, K. P., and Graves, L. J.: Follicular Conjunctivitis in School Children as Expression of Vitamin A Deficiency, Am. J. Dis. Child. **62**:101-114 (July) 1941.

21. Weiner, A. L., and Levin, A. A.: Pityriasis Rubra Pilaris of Familial Type: Experiences in Therapy with Carotene and Vitamin A, Arch. Dermat. & Syph. **48**:288-296 (Sept.) 1943.

22. Carleton, A., and Steven, D. M.: Keratosis Follicularis: Study of Four Cases, Arch. Dermat. & Syph. **48**:143-150 (Aug.) 1943. Stannus, H. S.: Proc. Roy. Soc. Med. **38**:337-342, 1945.

23. Peck, S. M.; Glick, A. W., and Chargin, L.: Vitamin A Studies in Cases of Ichthyosis, Arch. Dermat. & Syph. **48**:32-34 (July) 1943. Welton, D. G.: Keratosis Follicularis with Unusual Involvement of Scalp: Report of Case, ibid. **47**:398-404 (March) 1943.

24. Peck, S. M.; Glick, A. W.; Sobotka, H., and Chargin, L.: Vitamin A Studies in Cases of Keratosis Follicularis (Darier's Disease), Arch. Dermat. & Syph. **48**:17-31 (July) 1943.

25. Ruch, D. M.; Brunsting, L. A., and Osterberg, A. E.: Proc. Staff Meet., Mayo Clin. **21**:209-218, 1946.

CONCLUSION

The studies which are here reported show that the mechanism which regulates the plasma vitamin A in the human organism plays a role in various clinical conditions, such as hepatic diseases, fever, pregnancy and cutaneous disorders. Without question the liver plays an important role in the regulatory process controlling the amount of vitamin A circulating in the blood. But there are indications that other factors are also of importance. Knowledge of the regulation of the plasma vitamin A today is comparable to what was known of the regulation of blood sugar before insulin was discovered. In spite of well established facts which were discussed in this article, relative to the problem of the plasma vitamin A, investigations are still in a "preinsulin era," and much work must yet be done.

506 West Belden Avenue (14).

News and Comment

GENERAL NEWS

International Congress on Rheumatic Diseases.—The current interest in arthritis and the other rheumatic diseases received additional impetus when several hundred physicians from the United States and foreign countries gathered at the Waldorf Astoria Hotel, New York, for the seventh International Congress on Rheumatic Diseases, from May 30 to June 3, 1949. This congress was sponsored and supported by several organizations, including the American Rheumatism Association, the United States Public Health Service and the Arthritis and Rheumatism Foundation, the last being established in 1948.

A number of official delegates were appointed. Dr. W. S. C. Copeman, of London, England, who also spoke at the congress on Thursday, June 2, on fibrositis, was the delegate from Great Britain. Dr. Fernando H. Ramos, from Montevideo, was the official representative from Uruguay. He took part in the clinic session on "The Painful Shoulder," Friday, June 3. Dr. A. R. Moreno, of Buenos Aires, represented Argentina officially and presented a paper for the Sixth Plenary Session on Thursday, June 2, entitled "Possible Relations between Rheumatic Disease and Allergy: Its Experimental Study in the Rabbit." Many other distinguished visitors from these and other countries took part in the sessions. Physicians from the United States were also heavily represented on the program.

After the closing of the congress, most of the participants went to Atlantic City, N. J., for the annual session of the American Medical Association, which included several scientific exhibits on the rheumatic diseases and several papers read before various sections.

A postconvention tour covered the sixteen days following the session of the American Medical Association. Philadelphia, Boston, Buffalo, Detroit, Chicago, Rochester, Minn., and Washington, D. C., were the cities visited. Scientific sessions were held at Philadelphia, Boston and Rochester, Minn. Sightseeing with motor coaches was included in the itinerary for those visiting Boston, Washington, D. C., Niagara Falls, N. Y., and Chicago.

Children's Hospital, Washington, D. C., Forms Alumni Association.—The Children's Hospital, of Washington, D. C., has formed an alumni association with the following officers: president, Dr. Joseph S. Wall; vice president, Dr. Edgar P. Copeland, and secretary-treasurer, Dr. Archibald R. MacPherson.

Examinations of American Board of Pediatrics, Inc.—The American Board of Pediatrics, Inc., held written examinations under local monitors on Friday, June 24, 1949, from 2 to 4 p. m. The next oral examinations will be in Cleveland on Sept. 16, 17 and 18, 1949, in New York on Oct. 21, 22 and 23, 1949, and in Chicago on Dec. 9, 10 and 11, 1949.

Kappa Delta Award for Research in Orthopedic Surgery.—The National Council of the Kappa Delta Sorority has inaugurated a prize of $1,000 to be given annually by the American Academy of Orthopaedic Surgeons for the best research

in orthopedic surgery performed during the year by a person in the United States. The recipient of the first award, for the year 1949, will be announced at the seventeenth annual convention of the Academy, in New York, Feb. 11, 1950. Those wishing to compete for this prize may secure further information from Dr. Walter Stuck, 1426 Nix Professional Building, San Antonio, Texas, chairman of the award committee for 1949.

Convention of National Society for Crippled Children and Adults.—The annual convention of the National Society for Crippled Children and Adults will be held Nov. 7, 8 and 9, 1949, at the Commodore Hotel, New York.

NOTICES

Contributions for Medical Library of University of Chile Requested.—The National Committee for Chile is now receiving gifts for the library of the medical school of the University of Chile at its new collection center in the Library of Congress, Washington, D. C. The newer materials in the library, including periodicals, books and reference works, were totally destroyed in the recent fire. Medical periodicals of the last ten years and recent medical books are urgently needed. Your contribution will be appreciated. Please send it to the National Committee for Chile, Room 318, Library of Congress, Washington, D. C.

Abstracts from Current Literature

Metabolism; Infant Feeding; Milk and Other Foods

NUTRITIONAL DISORDERS IN INFANCY AND EARLY CHILDHOOD. A. ALTMANN, South African M. J. **21**:468 (July 12) 1947.

Altmann reviews briefly acute and chronic nutritional disorders from the point of view of causation, with a short description of the methods of investigation employed. These nutritional disorders he classifies as acute and chronic and separates them from the standpoint of etiologic origin into enteral infections, parenteral infections, alimentary factors, environmental factors and constitutional factors. The discussion is brief, well organized and well thought out.

McGUIGAN, Evanston, Ill.

FURTHER STUDIES OF HUMAN MILK. J. VAN ESPEN, Arch. franç de pédiat., **3**:289, 1946.

In his second extensive monograph on this subject, Van Espen accepts as criteria of the influence of fat on the nutrition of very young children certain classic signs of superalimentation, such as the curve for weight during the first month, the frequency of vomiting and the number of stools at the end of this period.

It was determined that the increase in weight directly paralleled the butter fat index. The frequency of vomiting was in direct proportion to the rise of fat, varying from 62 per cent among those fed breast milk, with a percentage of 3.3 to 4.6, to 29 per cent in infants whose food contained a percentage of 2 to 3.2 of butter fat.

The survey showed unexpected results in the report of stools, inasmuch as they averaged the same number in children fed milk either rich or poor in butter fat. In this protracted study, the evolution of the butter fat ratio during lactation was carefully followed and the remaining constituents of the milk given equal consideration. The influence of the mother's diet on the fat content and on the total volume secreted was thoroughly appraised. Exhaustive tables are attached.

AMESSE, Denver.

CARBOHYDRATE METABOLISM OF NEWBORN INFANTS. L. BARTA and A. SASS-KORTSÁK, Paediat. danub. **1**:88 (Feb.) 1947.

Eight to ten hours after birth, when the newborn infant was still starving, double dextrose tolerance tests were performed. In the majority of infants the Staub-Trangott effect could be observed. The tolerance for galactose was perfect in most of them. A determination of the capillary-venous surface performed during the dextrose tolerance tests proved that the presence of the Staub-Trangott effect, as well as its absence, is connected with hepatic function.

A comparison of the tolerance curves revealed that in healthy infants good dextrose assimilation may occasionally be associated with a decreased galactose tolerance, the reverse being, however, the more frequent.

The conclusion is drawn from the aforementioned observations that the characteristic carbohydrate metabolism of infants is due to the functional lability of the liver. The hepatic function is to be considered disturbed when there is intol-

erance for both dextrose and galactose, provided that either the breaking-up or the storing processes of carbohydrate are undisturbed. A diminished tolerance for one of these carbohydrates admits no conclusions concerning impaired hepatic function.

FROM THE AUTHORS' ABSTRACT.

Vitamins; Avitaminoses

RICKETS IN SINGAPORE. CICELY D. WILLIAMS, Arch. Dis. Childhood **21**:37 (March) 1946.

This is an interesting article on infant care and feeding and life in general in Singapore. The object of the paper is to prove the prevalence of rickets, a common disease that is responsible for a great deal of damage to child life, and to suggest its causes and the methods of combating the condition. This the author does in a most entertaining and interesting manner.

KELLY, Milwaukee.

A FATAL CASE OF BERIBERI IN A CHILD IN ENGLAND. E. C. ALLIBONE and H. S. BAAR, Arch. Dis. Childhood **21**:76 (June) 1946.

A fatal case of beriberi is described. The relationship of the myocardial changes to those observed in acute beriberi and in myocardial fibrosis is discussed.

KELLY, Milwaukee.

THE RELATIONSHIP OF THE BLOOD PYRUVIC ACID LEVEL TO DEFICIENCY STATES IN INFANCY AND CHILDHOOD. E. C. ALLIBONE and E. FINCH, Arch. Dis. Childhood **21**:165 (Sept.) 1946.

Normal resting levels for pyruvic acid in the blood of infants ranges from 0.43 to 1 mg. per hundred cubic centimeters, with a mean of 0.78 mg.

The presence of a toxic or infective state raises the blood pyruvic acid level, in spite of an estimated adequate thiamine intake.

The elevation of the pyruvic acid level is a valuable sign of a disturbance of carbohydrate metabolism but gives no indication of the cause.

The implication of the aforementioned observations is discussed.

KELLY, Milwaukee.

Hygiene; Growth and Nutrition; Public Health

OUTLINE OF THE CAUSES OF INFANTILE MORTALITY FOR TEACHING PURPOSES IN THE SOCIAL SERVICE. FRANCISCO JOSE MENCHACA, Arch. argent. de pediat. **26**:161 (Sept.) 1946.

Considering the necessity of preparing social workers efficiently in the fight against infant mortality, the author proposes a methodic classification of its causes. The outline will be useful not only as a didactic tool but as a guide for professional work as well.

ROJAS, Boston.

ACUTE ADRENAL INSUFFICIENCY IN THE NEWBORN. ALFREDO E. LARGUIA, J. R. VAZQUEZ AND J. D. VIDAL, Arch. argent. de pediat. **26**:180 (Sept.) 1946.

A clinical history of a case is presented. The following symptoms are pointed out as important in the early diagnosis of adrenal hemorrhage: sudden onset in a

2 to 4 day old baby; hyperthermia, polypnea, lividity of the skin and sometimes a generalized purpuric rash and convulsions; local and generalized symptoms related to the hemorrhage, and alterations in the blood chemistry (hemoconcentration, high nonprotein nitrogen level, hyponatremia, hypochloremia, hypoglycemia and hypercalcemia).

Differential diagnosis must be made among cerebral hemorrhage, acute pneumonopathies, septicemia and hemorrhagic renal infarct. From the pathogenic point of view, adrenal hemorrhages are directly related to trauma during delivery and to the involution of the adrenal cortex after birth. The onset of the disease coincides with the period of physiologic hypoprothrombinemia.

A scheme of treatment is included.

ROJAS, Boston.

DIFFICULTIES IN COMBATING HIGH INFANT MORTALITY. F. KREJSA, Pediat. listy **1**:85, 1946.

This paper is a contribution by a physician from a provincial town, in which he describes his efforts to lower the infant mortality rate in his district.

FROM THE AUTHOR'S SUMMARY.

COMPULSORY NURSERY ATTENDANCE FOR CHILDREN FROM THE AGE OF FIVE YEARS. MARIE SIBKOVA, Pediat. listy **1**:144, 1946.

The health and educational requirements of nurseries and their personnel are summed up, as a bill providing for compulsory nursery attendance for children from 5 years of age onward is under discussion.

FROM THE AUTHOR'S SUMMARY.

Prematurity and Congenital Deformities

THE STATISTICS OF PREMATURITY: A PLEA FOR STANDARDIZATION. J. L. HENDERSON, Arch. Dis. Childhood **21**:105 (June) 1946.

To enable accurate comparisons to be made among various methods of care and treatment, it is suggested that the following seven conditions provide a comparative basis:

1. A large number of infants
2. Accurate weighing at birth
3. Strict interpretation of the definition for "liveborn"
4. Inclusion of all liveborn infants with congenital malformations
5. Definition of the weight range of viable prematurity
6. Subdivision of the standard weight range into half-pound weight groups
7. Segregation of the infants of mothers who arrange early in pregnancy, without recommendation, to have their confinement in a hospital from those whose mothers' confinements in the hospital are classified as emergencies or are recommended because of ill health.

KELLY, Milwaukee.

LOSS OF HEAT AS THE CAUSE OF DEATH IN PREMATURE INFANTS. KORNEL CHMELO, Pediat. listy **1**:43, 1946.

Of a series of 585 premature infants, the cause of death in 27.5 per cent, i. e., in 161 of them, was loss of heat. The mortality rate was in direct proportion to the fall in temperature and to the degree of debility of the infants. A detailed survey is given. The mortality rate bore no relation to the season of the year. The author

hopes to lower the mortality rate in these infants to below 20 per cent by improving their general care and by preventing loss of heat.

FROM THE AUTHOR'S SUMMARY.

GENUINE MACROGENITOSOMIA PRECOX IN A GIRL THREE AND ONE-HALF YEARS OF AGE. KAMIL KUBAT, Pediat. listy **1**:62, 1946.

A case of macrogenitosomia precox is described. The clinical picture and an explanation of this particular type of sexual prematurity are included.

FROM THE AUTHOR'S SUMMARY.

Newborn

EPIDEMIC DIARRHEA OF THE NEWBORN. L. W. SAUER, Illinois M. J. **91**:201 (April) 1947.

In 1927, an epidemic of diarrhea occurred at The Cradle, Evanston, Ill., with a mortality of 32 per cent. After an exhaustive study of the epidemic, Dr. Gladys Dick devised an aseptic nursery technic as a preventive of further recurrence of infectious diarrhea.

During the eighteen years since the Dick individual aseptic nursery technic has been in use, several minor changes and short cuts have been introduced without the occurrence of any hand-borne cross infection. The technic is described.

BARBOUR, Peoria, Ill.

MEETING AN EMERGENCY SITUATION IN AN EPIDEMIC OF DIARRHEA AND ENTERITIS OF THE NEWBORN. E. V. THIEHOFF, Illinois M. J. **91**:254 (May) 1947.

The closing of the maternity floor of one of the three hospitals of Peoria, Ill., because of an epidemic of diarrhea of the newborn created an emergency problem. The problem was where the maternity patients who normally would be delivered on the closed floor of the hospital could be taken care of. The city health commissioner called a meeting of representatives of the various medical and social agencies involved in the solution of the emergency problem. The program planned and the results thereof are described in this article.

BARBOUR, Peoria, Ill.

PEMPHIGUS OF THE NEWBORN. J. NAMECHE, Acta pædiat. belg. **1**:29, 1946-1947.

After a historical review and discussion of nomenclature, the author describes first this disease group in general and then adds the experiences at the maternity station of the Saint Pierre Hospital in Brussels, Belgium. The clinical aspect is characterized by rapidly appearing bullae, encircled by reddened skin. The condition is acute and contagious. The bullae are filled at first with a serous fluid which becomes cloudy later. The tops of the bullae peel off, leaving red erosions. Crusts do not form on the skins of newborn infants but do on those of older children. The healed skin is often pigmented for several months. The localization is the hypogastric region, the groin and the thigh. The duration is one to two weeks. The patient's general condition is good and complications are occasional local progress of the infection.

A malignant form of pemphigus, or exfoliative dermatitis, shows easily peeling skin (Nikolsky's sign), with general symptoms of fever, loss of weight, coma and death. The causative agent is the staphylococcus. Streptococcic infection occurs only secondarily. The disease is seen in epidemic, sporadic or endemic form

(this last is most frequent). It is transferred by contact with attendants of the child, either directly or indirectly, through linen and other items. The possibility of hematogenous or intrapartum infection is discussed but rejected. For differential diagnosis the author considers syphilis, bullous strophulus, hematogenous dermatitis, dermatitis medicamentosa, epidermolysis bullosa, burns, infantile desquamative erythroderma (Leiner's disease) and ichthyosis. Strict aseptic procedures and supervision of all persons involved directly or indirectly in the attendance of the child are recommended as prophylactic measures. These are far superior to the therapeutic measures of cutaneous applications. As the latest in treatment, the author recommends administration of 100,000 units of penicillin in twenty-four hours and/or use of penicillin ointment. A statistic evaluation of the experiences at the Saint Pierre Hospital follows and leads to the conclusion that hygienic measures by reliable attendants are of utmost importance and have suffered during the war years. Vaccination against pemphigus remains without success.

GLASER, Chicago.

Acute Contagious Diseases

REPORT OF A CASE OF IRIDOCYCLITIS ASSOCIATED WITH CHICKENPOX. J. W. HALLETT, Am. J. Ophth. **29**:459 (April) 1946.

Hallett reports a case of iridocyclitis in a 7 year old boy three days after he contracted chickenpox. Although the attack was severe, the eye recovered fully.

W. S. REESE. [ARCH. OPHTH.]

THE RELATIONSHIP OF RUBELLA IN THE MOTHER TO CONGENITAL CATARACTS IN THE CHILD. E. L. GOAR and C. R. POTTS, Am. J. Ophth. **29**:566 (May) 1946.

Goar and Potts report 7 cases of congenital cataract, in all but 1 of which the mother had had rubella. Fair results in all were obtained by the Ziegler through and through discission.

W. S. REESE. [ARCH. OPHTH.]

THE EFFECT OF STILBESTROL ON RESISTANCE TO EXPERIMENTAL POLIOMYELITIS. F. J. CURLEY and W. L. AYCOCK, Endocrinology **39**:414 (Dec.) 1946.

The wide discrepancy between the pattern of distribution of the virus of poliomyelitis and that of the paralytic disease is an indication that added factors act as major determinants of the frank disease among those exposed to the virus.

Experiments are reported which serve to emphasize the set of conditions under which monkeys exhibit increased resistance to experimental poliomyelitis under estrogenic hormone as previously reported. Treatment of normal monkeys with stilbestrol produced no increase in resistance, while treated castrated monkeys showed a decided increase in resistance to intranasal instillations of the virus. Stilbestrol-treated animals which had resisted repeated intranasal instillations of virus, although kept on stilbestrol, showed no increased resistance to subsequent intracerebral inoculation.

JACOBSEN, Buffalo.

CUTANEOUS DIPHTHERIA. JACK V. CHAMBERS, U. S. Nav. M. Bull. **46**:744 (May) 1946.

A case of cutaneous diphtheria is reported in which the condition originated as a blister on a lower extremity. Examination a year after onset revealed a large

round ulcer, with raised, rolled edges of a bluish tinge, covered with a hard leathery scab. Smears and cultures revealed Corynebacterium diphtheriae. The fermentation reaction likewise was characteristic for this organism. Results of treatment with intramuscular and local application of diphtheria antitoxin were unsuccessful at the end of seventeen days. The ulcer healed rapidly after the injection of 600,000 units of penicillin over a six day period in conjunction with topical compresses.

The author suggests that antitoxin should always be administered, in spite of its failure to produce a cure, in order to counteract possible paralysis.

RODIN, South Bend, Ind. [ARCH. DERMAT. & SYPH.]

NICOTINIC ACID CONTENT OF THE SPINAL FLUID OF CHILDREN WITH POLIOMYELITIS. G. MURANO and V. BAFFI, Pediatria **55**:177 (April-June) 1947.

Analyses for nicotinic acid were made on the blood and spinal fluid of 27 patients with poliomyelitis, of 8 healthy children and of 15 patients with other pathologic involvement of the central nervous system or meninges. No unusual findings resulted from the determinations of the blood nicotinic acid. However, in all the spinal fluids from patients with acute poliomyelitis, i. e., up to twenty days after the onset of the disease, the nicotinic acid content was much higher (175 to 500 gamma per hundred cubic centimeters) than in the spinal fluids of the other patients and the normal children examined (36 to 60 gamma per hundred cubic centimeters).

The ratio $\frac{\text{Spinal fluid nicotinic acid}}{\text{Blood nicotinic acid}}$ in patients with acute poliomyelitis varied from 0.90 to 1.88, whereas in the other patients the ratio varied from 5.7 to 18.8. The ratio is offered as of diagnostic value in poliomyelitis. Sodium nicotinate, 55 to 66 mg., was given intrathecally to 7 children. A mild reaction with slight fever occurred in the healthy children, and a severe reaction with high fever in 2 of the children with acute poliomyelitis. There was no change in the blood. Ratios for nicotinic acid in the spinal fluid were unchanged following the injections. It had been hoped that some therapeutic reaction might follow these injections in patients with poliomyelitis, but the authors state they are unable to draw conclusions from the evidence obtained.

HIGGINS, Boston.

Acute Infectious Diseases

SEVERE PHAGEDENIC LESIONS OF TYPHOID ORIGIN IN A 4 YEAR OLD GIRL: REPORT OF A CASE. FLORENCIO BAZAN and E. SUJOY, Arch. argent. de pediat. **26**:186 (Sept.) 1946.

The clinical history is given. The lesions were located at the nasal septum, in the commissure of the lips, on the digits and in the umbilical region; the patient also presented necrotizing otitis and ulcerated tonsils.

A brief review of the literature on the subject is included. Several opinions by different authors on the causation and pathogenesis of the lesions are also mentioned.

The treatment used for this patient consisted of blood transfusions, antityphoid vaccine and injections of sulfarsphenamine (sulfarsenol®).

ROJAS, Boston.

TETANUS AND PENICILLIN. ALFREDO SEGERS and R. HIRIART, Arch. argent. de pediat. **26**:194 (Sept.) 1946.

A severe case of subacute tetanus which was not modified by massive administration of antitoxin is reported. Three days after his admission, the patient was

given penicillin and antitoxic serum. A remarkable improvement was noted after the first day of treatment, and the patient was discharged from the hospital ten days after his admission

ROJAS, Boston.

THE BACILLARY DYSENTERIES. J. SZURTRÉLY, Paediat. danub. **1**:168 (March) 1947.

A summary of the latest views on the disease, together with the results obtained from studies during the two years following the war, is given.

Diagnosis must be based primarily on clinical symptoms. If possible, the various types of the bacilli should be isolated. The role specific bacteriophages play in the culture is pointed out. Serologic reactions are of little value in diagnosis, as they are rarely positive before the tenth day of the disease, but agglutination tests made with the filtrate of the stools may reveal the disease as early as in the first week. The feces contain few bacilli but many polymorphonuclear leukocytes. The presence of macrophage cells is characteristic of bacillary dysentery.

Prognosis is greatly influenced by the age and state of the patient. In 1945, the bad hygienic conditions in Budapest, Hungary, led to a severe epidemic, the death rate of which was as much as 53.8 per cent. Of 147 seriously ill patients admitted to the pediatric department of the University of Budapest, only 44, that is 30 per cent, died. The figure was considered favorable as the patients, either artificially fed infants or undernourished children, were in poor condition, 22 of them displaying starvation edema. Toxicosis and convulsions were frequently observed in those in the acute stage. Eighty-eight per cent of the deaths occurred in infants under 1 year of age.

In addition to the generally known methods of control, bacteriophages may be of use in prophylaxis. In treatment, the best effects were seen after daily intramuscular injections of sulfathiazole in doses of 0.20 grain (12 mg.) per kilogram of body weight for infants. Repeated microtransfusions had an excellent effect.

In infancy, human milk is the diet of choice. Should this be unavailable, skimmed milk can be given. This is suitable also for children's diets.

FROM THE AUTHOR'S ABSTRACT.

Chronic Infectious Diseases

CONGENITAL TUBERCULOSIS. MARY R. HUGHESDON, Arch. Dis. Childhood **21**:121 (Sept.) 1946.

A review is made of the cases of congenital tuberculosis published between 1935 and 1945.

Four cases of congenital transplacental tuberculosis which occurred in a childrens' hospital in England between 1932 and 1944 are described. One patient died at the age of 12 days, the second at 48 days, the third at 13 months and the fourth at 27 months. In all 4 children there were primary complexes in both liver and lungs.

It is suggested that congenital tuberculosis may be commoner than has hitherto been supposed and may not be invariably fatal.

KELLY, Milwaukee.

MONGOL WITH ABSCESS IN NECK FROM TUBERCULOSIS OF CERVICAL SPINES. CHARLES DONALD, Proc. Roy. Soc. Med. **39**:587 (Aug.) 1946.

The patient at 2 years of age was noticed by the mother to have a lump in his neck which steadily increased in size, while the child lost weight. Physical examination showed the classic mongoloid appearance and a rounded, fluctuant mass in the postcervical region, lying under the skin and apparently attached to bone. Full movement of spine was present. The aspirated pus was thick and creamy, with no tubercle bacilli. The reaction to the Mantoux test was positive (1:1,000). A roentgenogram showed tuberculosis of the vertebrae (Pott's disease). The appearance of the chest was normal. Cerebrospinal fluid revealed no abnormality.

WILLIAMSON, New Orleans.

CLINICAL AND EXPERIMENTAL STUDY OF TORULOSIS. ROBERT DEBRÉ, MAURICE LAMY, C. LEBLOIS, JEAN NICK, GRUMBACH and E. NORMAND, Ann. pædiat. **168**:1 (Jan.) 1947.

The authors describe a case of torulosis in a child with malignant lymphogranulomatosis, confirmed by biopsy of a lymphatic gland and by the effect of roentgen therapy. Three weeks after resumption of normal life, headache, photophobia and a subcomatose state developed. A fist-sized ulcerating tumor appeared on the chin. Death occurred after two weeks.

In the spinal fluid yeastlike round to oval bodies without nuclei were discovered which were identified as Torula histolytica. The glands were transformed like those of Hodgkin's disease, and whitish, point-shaped structures were found on the pia mater. Changes in the spleen and liver as in lymphogranulomatosis were observed, as well as inflammatory nodules in nearly all the organs, which also showed the same bodies as the spinal fluid.

T. histolytica attacks the human organism through the skin and becomes localized in most of the internal organs, especially in the brain and the meninges. The symptoms may correspond to those of a cerebral tumor. The diagnosis is proved by the presence in the meninges of the organism, and, if the illness takes a more chronic course, in perivascular granulomas and cysts.

Guinea pigs and mice react to intraperitoneal inoculation with the development of typical lesions which lead to death within days or weeks.

Localized torulosis can be treated surgically. In the future penicillin should be tried for the generalized forms.

FROM THE AUTHORS' SUMMARY.

Diseases of Blood, Heart and Blood Vessels and Spleen

RETICULO-ENDOTHELIAL IMMUNE SERUM (REIS): V. AN EXPERIMENTAL ANEMIA IN BARTONELLA INFECTED RATS PRODUCED BY ANTI-BLOOD IMMUNE SERUM. C. M. POMERAT, EDWARD H. FRIEDEN and ENID YEAGER, J. of Infect. Dis. **80**:154 (March-April) 1947.

The historical background for the use of minute doses of antiorgan preparations for the stimulation of blood elements is reviewed. Additional evidence of the damaging cytotoxic effects of high concentrations of antiserums directed against the spleen, bone marrow and blood are presented.

An experimental anemia of the regenerative type having a remarkably uniform course is described as the result of a single injection of a homologous antiblood immune serum. This preparation apparently does not primarily damage the retic-

uloendothelial system, since the anemia produced is not accompanied with the bartonellosis, such as that induced by reticuloendothelial immune serum of the antispleen and bone marrow types or by splenectomy in rats having this infection in a latent state. The anemia-inducing factor is contained in the globulin fraction of such antiserums.

Fragmentary evidence is presented to suggest that antiblood immune serums may uncover latent bartonellosis when the reticuloendothelial system is affected by other experimental procedures.

TOOMEY, Cleveland.

THE ETIOLOGY AND THE EFFECTS OF MINOR GRADES OF ANAEMIA IN YOUNG CHILDREN: STUDIES AT A GROUP OF WAR-TIME DAY NURSERIES. HELEN M. M. MACKAY, R. H. DOBBS, KAITILIN BINGHAM and W. J. MARTIN, Arch. Dis. Childhood **21**:145 (Sept.) 1946.

An attempt has been made to determine some of the etiologic factors influencing the hemoglobin level of children attending eight wartime day nurseries in Islington, a borough in North London, England. The investigation lasted nearly two and a half years, and more than 900 children were examined.

There was a rise in mean hemoglobin level in the summer months, both in 1943 and 1944. The larger rise was in 1943 and was noted after the children's diet had been improved.

The diet provided at the nurseries, augmented with more rationed food at home, was, in the opinion of the authors, better than most children receive at home; hence it is probable that many children of preschool age living at home receive insufficient iron in their food. Since the morbidity rate was high, there is little doubt that infections depressed the mean hemoglobin level and retarded the full effect of iron therapy.

A 6 per cent rise in hemoglobin level produced by iron therapy was accompanied with a reduction in the absentee rate of about 26 per cent, compared with that of a control group in the same nursery, i. e., the degree of anemia present at this nursery was sufficient to lower resistance to infection, and resistance could be raised by giving iron. The anemia affecting the nursery children was largely a nutritional iron deficiency anemia responding to iron therapy. Other factors in the diet, for example, phytic acid, influenced iron absorption, and the high incidence of infections was also a factor of importance in the causation of the anemia.

KELLY, Milwaukee.

GAUCHER'S DISEASE. DONALD PATERSON, Proc. Roy. Soc. Med. **39**:587 (Aug.) 1946.

A case of Gaucher's disease was reported in a boy 7½ years old. The liver and spleen on examination were found to be enlarged down to the level of the umbilicus. Both were firm and hard, and the surface of the liver was irregular. No evidence of ascites or portal obstruction was present, and the general condition was good. Splenectomy and biopsy of the liver were performed. The reports of laboratory studies made immediately on the patient's admission and sixteen months later are given. Since operation, the liver has increased in size and has become more nodular.

WILLIAMSON, New Orleans.

Diseases of Nose, Throat and Ear

FOREIGN BODIES IN THE UPPER AIR PASSAGES: SPIKES OF GRAIN. FÉTHIYE AYRAL, Arch. franç. de pédiat. **3**:543, 1946.

From the Children's Division of the University Medical Department of Istanbul, Turkey, the author reports his observations on accidents of foreign bodies in the upper respiratory passages and compares his data with those from the Chevalier Jackson Clinic, in Philadelphia, where at least 80 per cent of the cases were shown to be preventable.

The reaction to foreign bodies in the respiratory corridor depends on various factors, which in turn determine in great measure the prognosis in any given case. Among these factors the author includes the position of the foreign body, the duration of the obstruction, the degree of closure, and the age and general health of the patient, as well as the nature of the obstructive agent.

Organic bodies are prone to produce the most serious effects and always indicate a graver prognosis. This is noted particularly following the aspiration of beans or peas. Among the commonest accidents, however, are those seen in infants and young children who have attempted to chew the beards of grain, especially of barley, oats or certain varieties of wheat. The spikes, or bristle-like awns, readily gain access to the respiratory passages and in a short time traverse the lungs and the pleura, to appear in a furuncle-like eruption on the wall of the chest. On extraction of the spike, the lesions promptly heal. Three personal cases are cited with full clinical histories.

AMESSE, Denver.

PRESUMED CONGENITAL METAPLASIA OF THE EPITHELIUM OF THE NASAL MEATUSES AS A CAUSE OF OZENA. L. V. RUSCA, Lattante **18**:276 (May) 1947.

The nasal tissue of 12 infants between 2 days and 1 year of age was studied histologically for the type of epithelium, number of glands and the amount of lymphoid tissue. The infants had died from various causes. No evidence was found to indicate a fundamental congenital condition which might later be the basis of ozena.

HIGGINS, Boston.

EFFECT OF PENICILLIN IN OTOLOGIC DISORDERS OF INFANTS AND CHILDREN. E. GYÖRGY, Ann. pædiat. **168**:75 (Feb.) 1947.

Penicillin was given in acute inflammations of the cavum tympani with extraordinarily good results. In infants and children with severe disorders of the lungs, no further otologic complications were observed in such inflammations when penicillin was administered.

In inflammations of the tympanum in which symptoms of bony changes had already become apparent, although operation could not be avoided through administration of penicillin, the time for healing of the wound was shortened. After operation for mastoiditis (antrotomy), the wound was closed, while systemic treatment with penicillin was continued. The wound healed per primam within six to ten days. In about the same time the tympanic discharge stopped.

FROM THE AUTHOR'S SUMMARY.

Book Reviews

Control of Pain in Childbirth: Anesthesia, Analgesia, Amnesia. Third edition, revised and enlarged. By Clifford B. Lull, M.D., and Robert A. Hingson, M.D. Price, $12. Pp. 522, with 169 illustrations. Philadelphia: J. B. Lippincott Company, 1948.

Since Sir James Simpson first made use of chloroform for the relief of pain in parturition, the lessening of suffering in delivery has been a subject of continual interest. Many methods of anesthesia and analgesia have appeared. Usually, when a new method of pain relief has been devised, there has been criticism of both its efficiency and its safety. Simpson himself met his full share of this. Today, the employment of some method for relief of pain in obstetric practice is generally accepted, although difference of opinion may be found as to the procedures to be used.

The authors of this book, which now appears in its third edition, have brought together a large amount of information, indeed, about all that is of real value, in the field of obstetric anesthesia and analgesia. While they are enthusiastic proponents of the use of caudal anesthesia, they cover the entire field of obstetric pain relief very well.

One may commend their approach to the discussion from the standpoint of basic science, for the two first chapters are devoted to a consideration of the anatomic structures involved in regional nerve block and spinal anesthesia and to a discussion of the pharmacologic aspects of the agents used in the induction of general anesthesia and amnesia.

A brief discussion of the psychologic methods for management of pregnancy and labor is included.

Caudal anesthesia receives more attention than any other one method. The technic, armamentarium and the regional anatomy which bear on the administration of caudal analgesia are discussed extensively. The history of pain relief in obstetrics is reviewed, and some of the methods which have been used and discarded are mentioned. The authors enlisted the cooperation of other writers in the provision of chapters on the material of which they might be particularly well informed.

Spinal anesthesia, which has of late assumed a more important position in the field of obstetrics, is discussed thoroughly and the technic is given. Excellent illustrations of the armamentarium and diagrams of areas rendered anesthetic by injections at the levels usually employed in obstetric practice are provided.

All the agents which have been used in inhalation anesthesia or analgesia are mentioned and those which are in present use are well discussed. In short, the book, while emphasizing the caudal method, gives an excellent review of the entire field of obstetric anesthesia and analgesia. The illustrations are excellent and of generous number. The book is of convenient size and printed on good paper; the printing itself is of excellent quality. The book may be recommended to those who desire a source of information on the various methods of obstetric pain relief and the technic of their use.

Directory of Pediatric Societies *

INTERNATIONAL

Sixth International Congress of Pediatrics

President: Prof. Guido Fanconi.
Secretary: Dr. H. Zellweger, Kinderklinik, Zurich.
American Committee:
President: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary-General: Dr. L. Emmett Holt Jr., 477 First Ave., New York 16.
Canadian Committee:
Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto.
Secretary: Dr. S. G. Ross, 1390 Sherbrooke St. W., Montreal.
Place: Zurich, Switzerland. Time: July 24-28, 1950.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

Pan American Congress of Pediatrics

President: Dr. Felix Hurtado, Habana, Cuba.

International Union for Child Welfare

President: Mrs. Gordon Morier.
Secretary: Dr. Georges Thélin, 43 Quay Wilson, Geneva, Switzerland.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

Associação Paulista de Medicina, Secção de Pediatrias

President: Dr. Armando de Arruda Sampaio.
First Secretary: Dr. Luis Fontoura y Lauro Machado Oliveira, Caixa Postal 2103, São Paulo, Brazil.
Second Secretary: Dr. Luiz Augusto Monteiro Toledo, Av. Brigadeiro Luiz Antonio 393, 1° Andar, São Paulo, Brazil.
Time: Twelfth of every month, 8:30 p. m.

Asociación Pediatrica de Guatemala

President: Dr. Carlos Manuel Monzon, Plazuela Santuario de Guadalupe, Guadalupe.
Secretary: Dr. Maria Isabel Escobar, Casa del Niño no. 1, Guadalupe.

British Paediatric Society

President: Dr. H. T. Ashby, 13 St. John St., Manchester, England.
Joint Secretary: Prof. Alan Moncrieff, Hospital for Sick Children, Great Ormond St., London W. C. 1.
Place: Windermere.

Bulgarian Pediatric Society

President: Dr. L. Ratchew, University Children's Hospital, Wassil Kolarow 46, Sofia.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Chinese Pediatric Society

President: Dr. W. S. Fu, The Shanghai Public Hospital for Children, 11 Hsiang Shan Road, Shanghai, China.
Secretary: Dr. T. F. Su, The Shanghai Public Hospital for Children, 11 Hsiang Shan Road, Shanghai, China.
Time: Concurrent with the annual conference of the Chinese Medical Association.

Czechoslovak Pediatric Society

Chairman: Prof. Dr. Jiri Brdlík, 2 Sokolská, Prague II.

Danish Pediatric Society

Chairman: Prof. Preben Plum.
Secretary: Dr. Elin Fog, Søllingsvey 17, Charlottenlund, Denmark.

German Pediatric Society

President: Dr. H. Kleinschmidt.
Secretary: Dr. F. Goebel, 5 Moorenstrasse, Düsseldorf, Germany (British Zone).
Place: Düsseldorf. Time: September 1949.

Hellenic Society of Pediatrics

President: Dr. C. Saroglou.
Secretary-General: Dr. S. Charokopos, Athens.

Indian Pediatric Society

President: Dr. K. C. Chaudhuri, 56/2 Creek Row, Calcutta 13.
Secretary: Dr. N. G. Mojumdar, 148 Russa Rd., Calcutta 26.
Place: 67 Dharamtala St., Calcutta 13. Time: Every second and fourth Wednesday of each month.

Nederlandsche Vereeninging voor Kindergeneeskunde

President: Dr. J. L. Keyzer, 114 St. Josephstr., Tilburg.
Secretary: Dr. H. Reerkink, % Sophia Kinderziekenhuis, 160 Gordelweg, Rotterdam.
Place: Children's Hospital or Pediatric Clinic. Time: Three times a year.

Palestine Jewish Medical Association, Section of Physicans of Children's Diseases

President: Prof. L. F. Meyer, 29 Toelsohn St., Tel Aviv.
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

Pediatric Society of New Zealand

President: Dr. Lawrence Ludbrook, The Lister Building, Victoria Street East, Auckland.

Royal Society of Medicine, Section for the Study of Diseases in Children

President: Prof. J. M. Smellie, 5 Flat, 8 Bolingbroke Grove, London S. W. 11.
Secretary: Dr. Mary Wilmers; Dr. R. Bonham Carter.
Place: 1 Wimpole St., London W. 1. Time: Fourth Friday of each month, 4:15 p. m.

Schweizerische Gesellschaft für Pädiatrie

President: Prof. Dr. A. Hottinger, Petersplatz 12, Basel.
Secretary: Dr. F. Hauser, Kinderspital, Basel.

Sociedad Boliviana de Pediatria

President: Dr. Carlos Ferrufino, Casilla 272, La Paz, Bolivia.
Secretary: Dr. Cecilio Abela D., Casilla 272, La Paz, Bolivia.
Place: S. C. I. S. P. Bldg. Time: Every Thursday.

Sociedad Chilena de Pediatria

President: Mr. Raúl Matte, Santiago, Chile.
General Secretary: Dr. Mariano Latorre, Santiago, Chile.
Place: Medical Society, Santiago, Chile. Time: 7:30 p. m., weekly.

SOCIEDAD CUBANA DE PEDIATRIA

President: Prof. Agustin Castellanos.
Secretary: Dr. Carlos Hernandez Miyares, Hospital Nuestra Señora de las Mercedes L y 21, Apartado 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Nuestra Señora de las Mercedes y Hospital Municipal de Infancia, Habana. Time: 9 p. m., last Wednesday of every month.

SOCIEDAD DOMINICANA DE PEDIATRIA

President: Dr. Alberto Peguero, Avenida Bolivar 117, Ciudad Trujillo, R.D.
Secretary-Treasurer: Dr. Jaime Jorge, Lea de Castro 19, Ciudad Trujillo, R.D.
Place: Children's Ramfis Hospital. Time: 8 p. m., last Friday of every month.

SOCIEDAD ECUATORIANA DE PEDIATRIA

President: Dr. Alfredo Ceballos Carrión.
Secretary: Dr. Rafael Compte Andrade, P. O. Box 3480, Guayaquil.
Place: Hospital Central. Time: 11:30 a. m., every Friday.

SOCIEDAD MEXICANA DE PEDIATRIA

President: Dr. Jesus Avarez de los Cobos.
Secretary: Dr. Luis Torregrosa F.
Place: Hospital Infantil. Time: Last Thursday of every month.

SOCIEDAD DE PEDIATRÍA DE CONCEPCIÓN (CHILE)

President: Dr. Raúl Ortega A., Hospital Clínico Regional, Concepción.
Secretary: Dr. Fidel Urrutia, Hospital Clínico Regional, Concepción.
Place: Hospital Clínico Regional, Concepción. Time: 7:30 p. m., second Tuesday of every month from April to December, inclusive.

SOCIEDAD DE PEDIATRÍA DE CÓRDOBA

President: Dr. Juan Francisco Herrera, Córdoba, Argentina.
Secretary: Dr. Albino F. Dottori, Dean Fumes 2000, Esq. Ocaña (N.), Córdoba, Argentina.
Place: Comite pro Defenso del Niño, Dean Fumes 2000, Córdoba, Argentina.
Time: Once a month.

SOCIEDAD DE PEDIATRIA DEL LITORAL

President: Dr. Jose E. Celoria, Hospital Centenario, Rosario, Argentina.
Secretary: Dr. Gloria Lovell, Hospital Centenario, Rosario, Argentina.

SOCIEDAD DE PEDIATRÍA DE SANTIAGO DE CUBA

President: Dr. Antonio Béguez César, Sagarra 107, Santiago de Cuba.
Secretary: Dra. Susana Texdó Vaillant, Padre Pico 57, Santiago de Cuba.
Place: Colegio Médico. Time: Last Tuesday of every month.

SOCIEDADE PORTUGUÊSA DE PEDIATRIA

President: Prof. Almeida Garrett.
Secretary-Generale: Dr. Cordeiro Ferreira, Hospital de D. Estefania, Lisbon.

SOCIEDAD VENEZOLANA DE PUERICULTURA Y PEDIATRIA

President: Dr. Pastor Oropeza, Caracas.
Secretary: Dr. Guillermo Flores, Chacin, Sur 17, no. 26-2, Caracas.
Place: Hospital Municipal de Niños, Caracas. Time: Once a month.

SOCIEDAD YUCATECA DE PEDIATRIA

President: Dr. José Lavalle, Yucatan, Mexico.
Secretary: Dr. Francisco Solís, Yucatan, Mexico.
Place: Hospital del Niño, Yucatan, Mexico.

SOCIÉTÉ DE PÉDIATRIE DE PARIS

President: Dr. G. Heuyer, 1 Ave. Emile Deschanel, Paris 7e, France.
Secretary: Dr. Maurice Lamy, 94 rue de Varenne, Paris 7e, France.
Place: Hôpital des Enfants Maladies, 149 Rue de Sèvres. Time: 4:30 p. m., third Thursday of every month.

SOCIETÉ POLONAISE DE PÉDIATRIE

President: Dr. Jan Bogdanowicz, Dzialdowska 1, Warsaw.
Secretary: Dr. Zofia Leimbach, Mieroslawskiego 9 m.6, Warsaw.

SOUTH AFRICAN PAEDIATRIC ASSOCIATION

Chairman: Dr. Basil Melle, 89 Lister Bldg., 195 Jeppe St., Johannesburg, South Africa.
Secretary-Treasurer: Dr. Seymour Heymann, 17 Lister Bldg., 195 Jeppe St., Johannesburg, South Africa.

SWEDISH ASSOCIATION OF PEDIATRICS

President: Prof. A. Lichtenstein, Crown Princess Louise's Children's Hospital, 30 Polhemsgata, Stockholm.
Secretary: Dr. K. A. Melin, Farjestadsvägen 16, Ängby.

SWEDISH MEDICAL SOCIETY, SECTION FOR PEDIATRICS AND SCHOOL HYGIENE

President: Dr. R. Hybbinette, Kommendorsgatan 3, Stockholm.
Secretary: Dr. B. Broman, Eriksbergsgatan 1A, Stockholm.
Place: Various clinics and hospitals in Stockholm and Uppsala. Time: Second Friday of each month (except summer).

URUGUAYAN SOCETY OF PEDIATRICS

President: Dr. Héctor C. Bazzano.
Secretary: Dr. José M. Portillo.
Place: Club Medico del Uruguay, Avenida Agraciada 1464 (Piso 13), Montevideo.

VIENNA PEDIATRIC SOCIETY

President: Prof. Dr. A. Solé, Karolinen-Kinderspital, Sobieskigasse 31, Vienna 9.
Secretary: Doz. Dr. Asperger, Universitäts-Kinderklinik, Lazarettgasse 14, Vienna 9.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY.
SECTION ON PEDIATRICS

Chairman: Dr. Woodruff L. Crawford, 321 W. State St., Rockford, Ill.
Secretary: Dr. Margaret Mary Nicholson, 1801 Eye St. N. W., Washington, D. C.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. Warren R. Sisson, 319 Longwood Ave., Boston 15.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.
Annual Meeting: Place: San Francisco. Time: Nov. 14-17, 1949.

AMERICAN HOSPITAL ASSOCIATION, MASSACHUSETTS HOSPITAL ASSOCIATION

Chairman: Dr. Frank Wing, Boston.
Secretary: Dr. W. Franklin Wood, McLean Hospital, Waverly 79, Mass.

AMERICAN PEDIATRIC SOCIETY

President: Dr. Valjean Cooke, 8700 Argyle Ave., St. Louis.
Secretary-Treasurer: Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. P. E. Williams, 193 James St. S., Hamilton, Ontario.
Secretary-Treasurer: Dr. R. L. Denton, 1615 Cedar Ave., Montreal.

SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. Francis F. Schwentker, 601 N. Broadway, Baltimore, Md.
Secretary-Treasurer: Dr. Robert Ward, Bellevue Hospital, New York 16.
Place: French Lick, Ind. Time: 1950.

SECTIONAL

Intermountain Pediatric Society

President: Dr. Spencer Snow, 75 S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. Esther Gross, 202 E. South Temple St., Salt Lake City.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

New England Pediatric Society

President: Dr. Howard W. Brayton, 179 Allyn Street, Hartford 3, Conn.
Secretary-Treasurer: Dr. Harry Shwachman, 300 Longwood Ave., Boston.
Place: Boston Medical Library. Time: Three meetings a year, occurring from September to May.

North Pacific Pediatric Society

President: Dr. Clarence L. Lyon, 407 Riverside Ave., Spokane 8, Wash.
Secretary: Dr. Aldis B. Johnson, 515 Cobb Bldg., Seattle, Wash.

Northwestern Pediatric Society

President: Dr. Paul Bancroft, 909 Sharp Bldg., Lincoln, Neb.
Secretary-Treasurer: Dr. Northrop Beach, 1009 Nicollet Ave., Minneapolis.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Rocky Mountain Pediatric Society

President: Dr. William D. Rothwell Jr., Republic Bldg., Denver.
Secretary: Dr. Edwin T. Williams, 1850 Gilpin St., Denver 6.
Place: Children's Hospital of the Colorado General Hospital. Time: Monthly from October to June.

Southern Medical Association, Section of Pediatrics

Chairman: Dr. Carroll M. Pounders, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. Samuel F. Ravenel, 371 N. Elm St., Greensboro, N. C.

STATE

Alabama Pediatric Society

President: Dr. W. A. Daniel Jr., 115 S. Union St., Montgomery 5, Ala.
Secretary-Treasurer: Dr. D. B. Monsky, 905 Norman Bridge Rd., Montgomery 6, Ala.
Place: Montgomery, Ala. Time: September 1949.

Arizona Pediatric Society

President: Dr. Carl A. Holmes, 1401 N. 7th Ave., Phoenix.
Secretary-Treasurer: Jacob M. Sobol, 607 Heard Bldg., Phoenix.
Place: Phoenix or Tucson. Time: On call.

Arkansas State Pediatric Association

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

California State Medical Society, Section on Pediatrics

Chairman: Dr. Alice Potter, 1418 Ninth Ave., San Francisco 22.
Secretary: Dr. Carl Erickson, 595 E. Colorado, Pasadena.

Florida State Pediatric Society

President: Dr. Edgar W. Stephens, 910 Harvey Bldg., West Palm Beach.
Secretary: Dr. Hugh A. Carithers, 2000 Park St., Jacksonville 4.
Place: Concurrent with state association convention and fall meeting.

Georgia Pediatric Society

President: Dr. Lee Bivings, 20 Fourth St. N. W., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.
Place: Atlanta. Time: At time of annual state meeting.

HEZEKIAH BEARDSLEY PEDIATRIC CLUB OF CONNECTICUT

President: Dr. H. L. F. Locke, 179 Allyn St., Hartford, Conn.
Secretary: Dr. L. W. Minor, 119 Main St., Middletown, Conn.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

Chairman: Dr. Eugene T. McEnery, 4458 W. Madison St., Chicago 24.
Secretary: Dr. George L. Drennen, Jacksonville, Ill.

INDIANA STATE PEDIATRIC SOCIETY

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Matthew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

IOWA PEDIATRIC SOCIETY

President: Dr. Charlotte Fisk, 1201 Equitable Bldg., Des Moines 9.
Secretary-Treasurer: Dr. James Dunn, Davenport Bank Bldg., Davenport.

LOUISIANA STATE PEDIATRIC SOCIETY

President: Dr. William C. Rivenbank, 1448 Louisiana Ave., New Orleans 15.
Secretary-Treasurer: Dr. Bertha Wexler, 3439 Prytania St., New Orleans 15.

MEDICAL SOCIETY OF THE STATE OF NEW YORK, PEDIATRIC SOCIETY

Chairman: Dr. Thurman B. Givan, 115 Remsen St., Brooklyn 2.
Secretary: Dr. Reginald A. Higgons, Port Chester.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA, PEDIATRIC SECTION

Chairman: Dr. Waldo E. Nelson, Temple University, Philadelphia 40.
Secretary: Dr. Carl C. Fischer, 100 W. Coulter St., Philadelphia 44.

MICHIGAN STATE MEDICAL SOCIETY, PEDIATRIC SECTION

Chairman: Dr. H. J. Lewis, 2956 Biddle Ave., Wyandotte.
Secretary: Dr. Robert H. Trimby, 122 W. Hillsdale, Lansing.

MISSISSIPPI STATE PEDIATRIC SOCIETY

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Place, Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

NEBRASKA PEDIATRIC SOCIETY

President: Dr. George Clark, 1817 Vinton St., Omaha.
Secretary-Treasurer: Dr. Michael Crofoot, 1407 Medical Arts Bldg., Omaha 2.
Place: Children's Memorial Hospital. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

NEW HAMPSHIRE PEDIATRIC SOCIETY

President: Dr. Thomas B. Walker, Porthmouth.
Secretary-Treasurer: Dr. Ursula G. Sanders, Concord.

NEW JERSEY MEDICAL SOCIETY, PEDIATRIC SECTION

Chairman: Dr. Israel J. Wolf, 231 E. 31st St., Paterson.
Secretary: Dr. N. T. Crane, 147 E. 7th St., Plainfield, N. J.

NEW MEXICO STATE PEDIATRIC SOCIETY

President: Dr. M. K. Wylder, 221 W. Central Ave., Albuquerque.
Secretary-Treasurer: Dr. Ly Werner, 221 W. Central Ave., Albuquerque.

NORTH CAROLINA PEDIATRIC SOCIETY

President: Dr. Samuel F. Ravenel, 371 N. Elm St., Greensboro, N. C.
Secretary: Dr. Charles H. Gay, 1523 Elizabeth Ave., Charlotte, N. C.

NORTHERN CALIFORNIA PEDIATRIC SOCIETY

President: Dr. William C. Deamer, 3 Fifth Ave., San Francisco 18.
Secretary: Dr. A. L. Gleason, 1066 Longridge Rd., Oakland 10.
Place: San Francisco. Time: Second Thursday of February, April, September and November.

OKLAHOMA STATE PEDIATRIC SOCIETY

President: Dr. J. B. Snow, 625 N. W. 10th St., Oklahoma City.
Secretary: Dr. Bertha Levy, 1200 N. Walker St., Oklahoma City.
Place: With Oklahoma State Medical Association. Time: Annual Meeting.

PHILIPPINE PEDIATRIC SOCIETY

President: Dr. Alberto V. Tupas, Philippine General Hospital, Manila, Philippines.
Secretary: Dr. Artemio P. Jongco, Philippine General Hospital, Manila, Philippines.
Place: Philippine General Hospital. Time: Every two months.

SOUTH CAROLINA PEDIATRIC SOCIETY

President: Dr. B. Owen Ravenel, 95 Rutledge Ave., Charleston 16.
Secretary-Treasurer: Dr. John R. Harvin, 206 N. Sumter St., Sumter.

TENNESSEE PEDIATRIC SOCIETY

President: Dr. W. O. Vaughan, 2103 Hayes St., Nashville 5.
Secretary-Treasurer: Dr. C. Barton Etter, 1678 Peach, Memphis 12.
Place: Chattanooga. Time: Second Monday in April.

TEXAS PEDIATRIC SOCIETY

President: Dr. John K. Glen, 4412 Montrose St., Houston.
Secretary-Treasurer: Dr. James N. Walker, 3616 Tulsa Way, Fort Worth 7.
Place: San Antonio. Time: Oct. 7-8, 1949.

VIRGINIA PEDIATRIC SOCIETY

President: Dr. C. P. Brown, 142 W. York St., Norfolk.
Secretary: Dr. E. Berkeley Neal, 303 Washington Ave. S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

President: Dr. Thomas G. Folsom, 1139 Fourth Ave., Huntington.
Secretary: Dr. Warren J. Parsons, 425 Eleventh St., Huntington.

WISCONSIN STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

Chairman: Dr. M. G. Peterman, 411 E. Mason St., Milwaukee 2.
Secretary: Dr. L. M. Simonson, 1001 N. 8th St., Sheboygan.

LOCAL

ACADEMY OF MEDICINE OF CLEVELAND, PEDIATRIC SECTION

Chairman: Dr. John Hart Davis, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. Francis F. Silver, 9400 Euclid Ave., Cleveland.
Place: Allen Memorial Medical Library. Time: 8:15 p. m., October, December, February and April.

ACADEMY OF MEDICINE, TORONTO, SECTION OF PEDIATRICS

President: Dr. A. L. Chute, 170 St. George St., Toronto, Canada.
Secretary: Dr. John Fletcher, 262 St. Clair St. W., Toronto, Canada.
Place: Academy of Medicine. Time: 8:30 p. m., third Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, PEDIATRIC SECTION

Chairman: Dr. Charles O'Donovan Jr., 5 E. Read St., Baltimore.
Secretary: Dr. T. Campbell Goodwin, 16 E. Biddle St., Baltimore.

BRONX PEDIATRIC SOCIETY

President: Dr. Harry S. Pizer, 1726 Davidson Ave., New York.
Secretary: Dr. William A. Schonfeld, 211 W. 106th St., New York 25.
Place: Morrisania City Hospital. Time: 8:30 p. m., second Wednesday of each month, except June, July, August and September.

BROOKLYN ACADEMY OF PEDIATRICS

President: Dr. Walter R. Coles, 604–2d St., Brooklyn.
Secretary: Dr. Bernard Benjamin, 845 Park Pl., Brooklyn 16.
Place: Granada Hotel. Time: 8:45 p. m., fourth Wednesday of February, March, April, October and November.

Buffalo Pediatric Society

President: Dr. Douglas P. Arnold, 159 Bidwell Parkway, Buffalo, N. Y.
Secretary: Dr. Richard A. Downey, 786 Forest Ave., Buffalo 9, N. Y.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p. m., first Monday of each month from September to June.

Central New York Pediatric Club

President: Dr. T. Wood Clarke, 7 Cottage Place, Utica.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Third Tuesday of April and October.

Chicago Pediatric Society

President: Dr. Arthur F. Abt, 104 S. Michigan Blvd., Chicago 3.
Secretary: Dr. Alfred S. Traisman, 6349 N. Clark St., Chicago.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May inclusive.

Cincinnati Pediatric Society

President: Dr. John H. Hunt, Kemper Lane Bldg., Cincinnati.
Secretary: Dr. William F. Hunting, 6048 Montgomery, Cincinnati.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Dallas Pediatric Society

President: Dr. Guy Hacker, 5711 Goliad St., Dallas.
Secretary-Treasurer: Dr. George R. Branch, 4325 Cole Ave., Dallas.
Place: Children's Hospital of Texas and Bradford Memorial Hospital. Time: 1 p. m., third Saturday and 8:30 p. m., the first Tuesday of each month.

Detroit Pediatric Society

President: Dr. Harold B. Rothbart, 773 Fisher Bldg., Detroit 2.
Secretary: Dr. S. S. Bernstein, 650 Maccabees Bldg., Detroit.
Place: Variable. Time: Five times annually.

Fort Worth Pediatric Society

President: Dr. Frank Cohen, 712 S. Henderson, Fort Worth, Texas.
Secretary: Dr. H. H. Womack, 1422 Pennsylvania, Fort Worth, Texas.
Place: Welch and West Restaurant. Time: Third Friday of each month.

Honolulu Pediatric Society

President: Dr. Donald Marshall, Medical Group, Honolulu.
Secretary-Treasurer: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu

Houston Pediatric Society

President: Dr. Edward O. Fitch, 3720 Fannin St., Houston, Texas.
Secretary: Dr. Thomas J. Donovan, 3506 Sunset Blvd., Houston 5, Texas.
Place: River Oaks Country Club. Time: 7:30 p. m., last Tuesday of each month.

Kansas City (Missouri) Pediatric Society

President: Dr. Harry M. Gilkey, 1624 Professional Bldg., Kansas City.
Secretary: Dr. Leland Speer, 1022 Hoel Parkway, Kansas City.
Place: Kansas City General Hospital. Time: On call.

Knoxville-Oak Ridge Pediatric Society

President: Dr. Henry S. Christian, 205 Medical Arts Bldg., Knoxville, Tenn.
Secretary: Dr. John Eggleston, Oak Ridge Hospital, Oak Ridge, Tenn.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Elena Boder, 1830½ Lucille Ave., Los Angeles 26.
Secretary-Treasurer: Dr. Robert Simonds, 267 S. Beverly Dr., Beverly Hills, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: Second Monday of February, April, June, October and December.

MEDICAL SOCIETY OF THE COUNTY OF KINGS AND THE ACADEMY OF MEDICINE OF BROOKLYN, PEDIATRIC SECTION

President: Dr. Samuel Silber, 865 Park Place, Brooklyn.
Secretary: Dr. Jacob J. Lichterman, 1433 Union St., Brooklyn 13.
Place: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., fourth Monday of each month.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON PEDIATRICS

President: Dr. John Washington, 2032 Belmont Rd. N.W., Washington, D. C.
Secretary-Treasurer: Dr. George Maksim, 1418 Good Hope Rd. S. E., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N.W. Time: Spring and fall.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. C. Barton Etter, 1678 Peach, Memphis 12, Tenn.
Secretary-Treasurer: Dr. Jacob Danciger, 188 S. Bellevue Blvd., Memphis 4, Tenn.
Place: University Center, 4 S. Dunlap. Time: 7 p. m., second Tuesday of every month.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. James Conway, 1721 E. Lake Bluff Blvd., Milwaukee 11, Wis.
Secretary-Treasurer: Dr. Walter Polacheck, 425 E. Wisconsin Ave., Milwaukee 2, Wis.
Place: City Club of Milwaukee. Time: Second Wednesday in February, April, June, October and December.

NASHVILLE PEDIATRIC SOCIETY

President: Dr. John M. Lee, Doctors Bldg., Nashville, Tenn.
Secretary: Dr. Dan S. Sanders Jr., 2103 Hayes St., Nashville, Tenn.
Time: Third Thursday of every month.

NEW YORK ACADEMY OF MEDICINE, SECTION ON PEDIATRICS

Chairman: Dr. John Dorsey Craig, 120 E. 75th St., New York 21.
Secretary: Dr. Morris Greenberg, 143 W. 87th St., New York.
Place: New York Academy of Medicine, 2 E. 103rd St. Time: Second Thursday of each month from November to May, inclusive, 8:30 p. m.

OKLAHOMA CITY PEDIATRIC SOCIETY

President: Dr. Bertha M. Levy, 717 N. W. 22d St., Oklahoma City 3.
Secretary: Dr. C. M. Bielstein, 301 N. W. 12th St., Oklahoma City 3.
Place: Oklahoma Club. Time: On call.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Aims C. McGuinness, 1740 Bainbridge St., Philadelphia 46.
Secretary: Dr. F. H. Harvie, 133 S. 36th St., Philadelphia 4.
Place: College of Physicians, 19 S. 22nd St. Time: 8:30 p. m., second Tuesday from October to May, inclusive.

PITTSBURGH PEDIATRIC SOCIETY

President: Dr. J. A. Gilmartin, 3708–15th Ave., Pittsburgh.
Secretary-Treasurer: Dr. C. J. Stoecklein, Medical Arts Bldg., Pittsburgh 13.
Place: Municipal Hospital, Pittsburgh. Time: Second Friday in October, December, February and April.

QUEENS PEDIATRIC SOCIETY

President: Dr. Henry A. Reisman, 88-03 146th St., Jamaica, L. I.
Secretary: Dr. Moe Goldstein III, 3a 76th Ave., Forest Hills, L. I.
Place: Forest Hills, N. Y. Time: Third Monday of October, February, April; first Monday of December.

RICHMOND PEDIATRIC SOCIETY

President: Dr. Thomas S. Chalkley, 421 W. Grace St., Richmond 20, Va.
Secretary-Treasurer: Dr. John Paul Jones, 810 W. Franklin St., Richmond 20, Va.
Place: Medical College Hospital. Time: 8 p. m., third Thursday of each month, except June, July and August.

ROCHESTER PEDIATRIC SOCIETY

President: Dr. Jerome Glaser, 85 San Gabriel Dr., Rochester 10, N. Y.
Secretary-Treasurer: Dr. Daniel B. Peeler, 1162 Lake Ave., Rochester 13, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee.
Time: Third Friday of each month from October to May.

ST. LOUIS PEDIATRIC SOCIETY

President: Dr. Merl J. Carson, 500 S. Kingshighway, St. Louis.
Secretary-Treasurer: Dr. Jackson K. Eto, 734 Missouri Theatre Bldg., St. Louis 3.
Place: Hotel Branscome. Time: Second Thursday of each month from October to May, inclusive, 6:30 p. m.

SAN ANTONIO PEDIATRIC SOCIETY

President: Dr. Lucius Hill, 328 Medical Arts Bldg., San Antonio 5.
Secretary-Treasurer: Dr. Edith M. Bonnet, 228 Medical Arts Bldg., San Antonio 5.
Place: Bexar County Medical Library. Time: 7:30 p. m., first Tuesday of every month.

SEATTLE PEDIATRIC SOCIETY

President: Dr. Sherod M. Billington, Medical Dental Bldg., Seattle 1.
Secretary: Dr. Jack M. Doctor, 738 Broadway, Seattle 22.
Place: College Club. Time: Fourth Friday of each month from September to June at 6:30 p. m.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Harry F. Dietrich, 416 N. Bedford Dr., Beverly Hills, Calif.
Secretary: Dr. Clement H. Maloney, Beverly Hills, Calif.
Place: University Club of Los Angeles. Time: 6:30 p. m., second Wednesday in January, March, May, September and November.

UNIVERSITY OF MICHIGAN PEDIATRIC AND INFECTIOUS DISEASE SOCIETY

President: Dr. Ernest H. Watson, University Hospital, Ann Arbor.
Secretary: Dr. Harry A. Towsley, 1313 E. Ann St., Ann Arbor.

WACO PEDIATRIC SOCIETY

President: Dr. M. C. Carlisle, 1410 Austin Ave., Waco, Texas.
Secretary: Dr. F. W. Hoehn, 1422 Austin Ave., Waco, Texas.
Place: Provident Hospital, 1725 Colcord, Waco, Texas. Time: Third Thursday of each month.

WESTCHESTER COUNTY MEDICAL SOCIETY, PEDIATRICS SECTION (NEW YORK)

President: Dr. Harry W. Kaessler, 147 Prospect Ave., Mount Vernon, N. Y.
Secretary-Treasurer: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.
Place: The Roger Smith Hotel, White Plains, N. Y. Time: Fourth Thursday of every other month from September to June (dinner meetings).

WICHITA PEDIATRIC SOCIETY

President: Dr. Walter L. Schafer, 401 N. Emporia St., Wichita 2, Kan.
Secretary: Dr. William F. McGuire, 401 N. Emporia St., Wichita 2, Kan.
Place: Variable. Time: First Thursday of each month.

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